The Journal of Thoracic Surgery

VOL. 25 FEBRUARY, 1953 NO. 2

Original Communications

THE PATENT DUCTUS

A REVIEW OF SEVENTY-FIVE CASES WITH SURGICAL TREATMENT INCLUDING AN ANEURYSM OF THE DUCTUS AND ONE OF THE PULMONARY ARTERY

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IT MAY be categorically stated that a ductus arteriosus persistently patent after the fifth year of life, with or without symptoms, should be closed to avoid certain potential hazards. These are retardation in physical and perhaps mental development, cardiac dilatation and decompensation, and bacterial endocarditis and endarteritis. Should any of these complications appear before the age of 5, immediate operative closure of the ductus is indicated.[1] One infant in our series was operated upon for cardiac failure at the age of 16 months, and we have just operated upon a second infant 5 months old in incipient cardiac failure with a pulse rate of 160 to 180, a respiratory rate of 80 to 100, and a blood pressure of 110/0. Another lesion may well be present but definite improvement has occurred as shown by a present blood pressure of 100/68. On the other hand, Josefson[2] reported a patient who reached the age of 66 years before any harmful effects of the open ductus appeared. A woman of 79 died recently at San Francisco Hospital who was known to have had the murmur of an open ductus for many years, with cardiac symptoms for only about five years. At autopsy, a "small" patent ductus with great dilatation of the pulmonary artery and generalized cardiac hypertrophy were found. Why should there be this absence or long delay in the development of symptoms? Why in Maude Abbott's series[3] of ninety-two patients with patent ductus should 43 per cent die of cardiac failure at an average age of 24 years? In another series,[4] 71 per cent of the patients died before the age of 40, largely of cardiac complications. Writers generally speak in vague terms of "an increased burden on the heart as the patient grows older, which manifests itself by cardiac enlargement." Others emphasize only the size of the ductus as the determining

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery at Dallas, Texas, May 8-10, 1952.

factor in the development of cardiac symptoms. A sounder and more accurate explanation may be found in the abnormal physiology of the patent ductus, which is comparable in many respects with the pathologic physiology of a peripheral arteriovenous fistula.[5] The ultimate effect of a large arteriovenous fistula is cardiac dilatation, decompensation, and death if not closed by operation. These effects are dependent upon the deflection of a large volume of blood from the arterial bed with its high pressure directly into the capacious venous bed with its low or even negative pressure.[6, 7] Because of the low or absent resistance at the site of the fistula as compared with the capillary bed elsewhere, blood flows more freely through the fistula into the venous bed and back to the heart, thus increasing the cardiac output by the volume of blood flowing through the fistula and, in effect, engrafting upon the normal circulation a shorter parasitic circulation.[8]

The volume of blood deflected through the fistula is controlled by the size of the fistula, by its distensibility, and by distensibility of the vessels leading to and from the fistula. Given distensible vessel walls with compressible tissues in which these vessels lie, and an absence of fibrosis at the site of the fistula, the volume of blood flowing toward and through the fistula with its low resistance will tend always to increase. The result is a progressive dilatation of that part of the circulatory bed through which the short-circuited blood flows, including the artery and vein leading to and from the fistula, both sides of the heart, and the fistula itself. Recorded cases of peripheral fistulas indicate that the size of the fistula does not remain stationary, but may increase from 5 to 20 mm. in diameter over a period of 20 to 25 years. As the fistula dilates and the volume of short-circuited blood increases, all chambers of the heart become dilated, and decompensation is imminent and inevitable if the fistula is not closed. In the growing animal,[9] experimentally produced peripheral fistulas have resulted in remarkably large hearts characterized both by great dilatation and by great hypertrophy. It was suggested that when dilatation outstrips hypertrophy, as might be expected in the adult animal, decompensation is an early sequence, but when increasing hypertrophy parallels and is commensurate with the increasing dilatation, as might occur in a growing animal, great cardiac enlargement is possible without decompensation.

A patent ductus produces comparable effects upon the circulation with certain unique modifications: in intrauterine life, the right heart pumps blood through the ductus from the pulmonary artery to aorta against the peripheral resistance of the systemic circulation. In the fetus the circulating blood is about equally divided in volume flow between the right and left hearts. Up to birth, therefore, the two ventricles have been propelling forward equal volumes of blood against equal peripheral resistances with the result that normally at birth the two ventricles are equally developed, and both contribute equally to the formation of the apex of the heart.[6] Immediately after birth if, for some still unknown reason, the ductus remains open, the large reservoir of low resistance in the pulmonary bed attracts blood to it from the systemic circulation and causes a reversal of flow through the ductus which then trans-

ports blood from aorta to pulmonary artery (Fig. 1). It is apparent at once that under these circumstances the blood flowing through the ductus will flow only through the pulmonary artery, the lungs, the pulmonary veins, the left heart, and the aorta, completely by-passing the systemic circulation, the vena cava, and the right heart. The normal cardiac output of the left ventricle is thus greatly augmented by the volume of blood flowing through the ductus

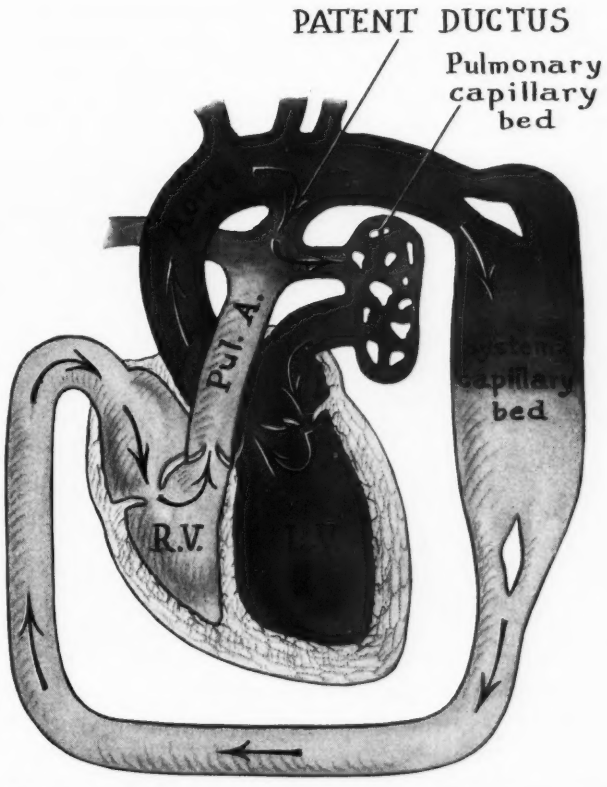

Fig. 1.—Schema of circulation in presence of a patent ductus transporting blood from aorta to pulmonary artery, resulting in dilatation of the left heart. The low resistance in the pulmonary arterial bed will continue to attract an increasing volume of blood through the ductus, resulting in its dilatation and in a progressive impairment of the circulation.

back to the heart. This short-circuited blood serves no purpose and may be considered parasitic upon the normal circulation. Eppinger and Burwell[10] demonstrated in a patient with a ductus that 25 liters of blood per minute flowed through the ductus and the left heart, and only 5 liters per minute flowed through the right heart. Closure of the ductus equalized the flow through the two ventricles to a little over 5 liters. The inevitable consequence of this greater volume flow of blood through the shorter circuit is a dilated left heart. The right heart, through which the smaller or normal volume of

blood flows, remains small. Indeed, the volume of blood flowing through the systemic circulation may be so reduced as to interfere with proper mental and physical growth. A number of our young patients blossomed in vigor and growth promptly after closure of the ductus, and one of two children (Fig. 2) who showed mental retardation in addition to other defects was apparently improved after elimination of the ductus. The literature contains other reports of the association of patent ductus with mental inadequacy.[5]

These pathologic effects of the abnormally patent ductus depend upon the low resistance to the flow of blood encountered in the pulmonary circulation, as compared with the high resistance to the flow of blood through the systemic capillary bed. Since a difference in the resistances of the two systems usually persists throughout life, and since, in consequence, the aortic pressure is

Fig. 2.—Two children with unilateral cataract, strabismus, some mental impairment, and patent ductus. (Each mother had rubella at one month of gestation). Ligation of ductus has resulted in definite improvement in mental status of child *a* and apparent improvement in child *b*.

permanently greater than pulmonic pressure, the tendency will be for blood to be deflected in increasing volume to the bed of less resistance. The effect of this increasing volume flow is progressive dilatation of that part of the circulatory bed through which the blood flows, including the ductus, and the left heart. Certain factors may limit or even prevent this increasing volume flow through the ductus, in which case the effects of the ductus will remain stationary throughout life. These factors are the resistance to dilatation inherent in the fibrous wall of the ductus itself, and the increasing fibrosis in the pulmonary capillary bed subjected for a prolonged period to the high arterial pressure of the systemic circulation.

The failure of a ductus to close completely may perhaps be due to a deficiency in fibrosis in the structure of its walls which, instead of contract-

ing normally after birth remain easily distensible, a characteristic which may persist throughout life, permanently preventing its automatic closure once it has provided a pathway to the reservoir of low resistance in the pulmonary bed. Indeed, failure to close in the first instance may be a direct result of the very low resistance in the pulmonary bed which attracts blood to it through the ductus. A flow once established would be an effective factor in maintaining the patency of the ductus, particularly if the differential in the resistances of the two systems is high. With the passage of time the difference in these resistances, though significant and effective, may lessen due to this factor of increasing fibrosis, and the tendency for the volume flow of blood through the ductus to increase is, therefore, correspondingly lessened. Hence, the disabling effects of the ductus and of the parasitic circulation which it produces, are slow in developing, and may be delayed for years, but they are nonetheless inevitable if the ductus remains patent, if its walls remain easily distensible, and if progressive dilatation of the ductus is not prevented by its inherent fibrosis or by the fibrosis of inflammation and infection. Just as a peripheral fistula, if large, may produce cardiac failure in three weeks, or if small may remain comparatively symptomless for many years and then end in cardiac decompensation because of progressive dilatation of the fistula, so may the harmful consequences of a patent ductus appear soon after birth or be delayed for many years. The actual course of events is determined by the degree of difference of resistances in the pulmonary and systemic beds, which in turn determines the degree of difference in the aortic and pulmonic pressures, and by the size, distensibility, and progressive dilatation of the ductus itself.

Certain authors contend that surgical ligation is indicated for the uncompensated patent ductus at whatever age signs of circulatory embarrassment appear, but for patients with a "compensated" ductus a policy of watchful waiting is advised. Our view is that since the open ductus provides a permanent passageway from a bed of high resistance to a bed of low resistance, it will never be fully "compensated," and will always tend to transport more and more blood within the limits of its distensibility or until the resistances in the two beds are more or less equalized. It is this concept of the pathologic physiology of a patent ductus that justifies the categorical statement that a ductus must be closed, preferably early in childhood, before the hazards of impairment of physical growth and cardiac decompensation arise.

The peripheral fistula and the patent ductus are also quite comparable in their effect upon blood volume and blood pressure.[11] With the opening of an experimentally produced peripheral fistula, there occurs a prompt fall in blood pressure which is compensated for by an increase in total blood volume commensurate with the fall in pressure and with the volume of short-circuited blood. If the fistula is small, the systolic and diastolic pressures may be restored to normal by this increase in blood volume. If the fistula is large, the diastolic pressure is permanently lowered due to the significant decrease in peripheral resistance at the fistula. When a peripheral fistula is closed by operation, there is a reversal of these changes (Fig. 3, *A*)—the systolic and

diastolic pressures are temporarily elevated but fall again as the previously increased blood volume returns to normal. The elimination of a peripheral fistula in each of three patients caused a reduction in total blood volume of 2,700, 1,000, and 1,200 c.c., respectively,[12] If a fistula is large, its closure will produce a permanent elevation of diastolic pressure due to the significant increase in peripheral resistance.

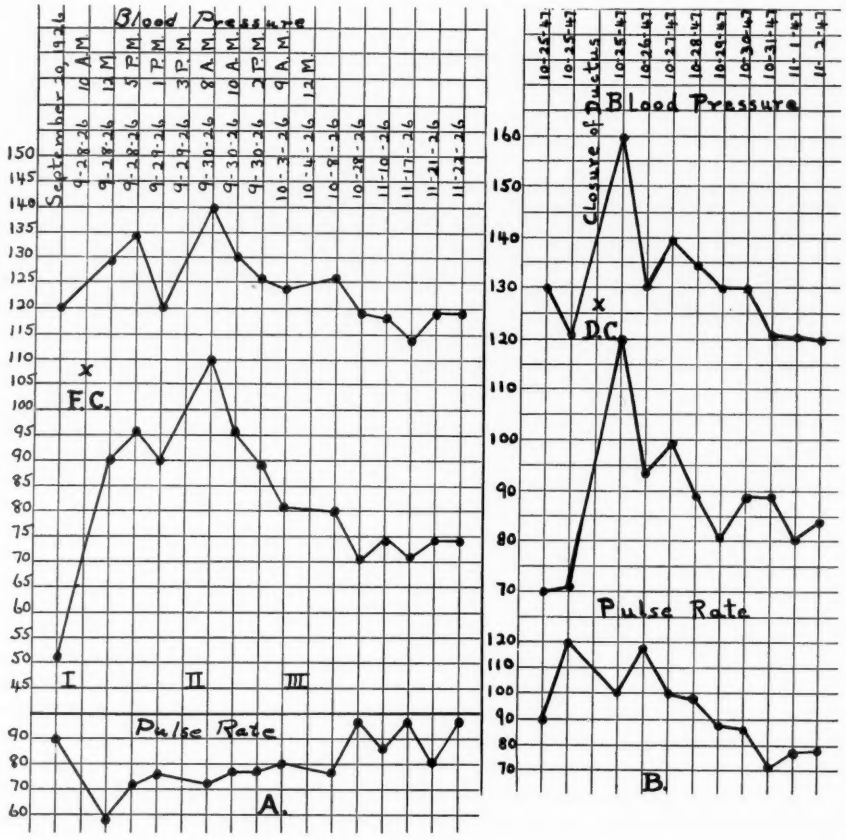

Fig. 3.—Similarity of blood pressure response to closure of fistula (Chart *A*), and closure of ductus (Chart *B*): The temporary abnormally high elevation of diastolic and systolic levels is due to temporary overdistention of the circulatory bed by the increase in total blood volume, which accompanies both these lesions. Diastolic pressure is permanently elevated due to increase in peripheral resistance.

Similarly, the production of a patent ductus by anastomosis of the left subclavian artery to the left pulmonary artery, as in the Blalock procedure, causes a fall in blood pressure, chiefly diastolic. That the blood volume is increased in the presence of a ductus was demonstrated by Eppinger, Burwell, and Gross,[13] who showed a reduction in blood volume following closure of a ductus in one case from 3,980 to 3,280 c.c., and in a second case from 4,130 to 3,720 c.c.

Closure of a patent ductus by operation is accompanied by a temporary abnormal increase both in systolic and diastolic pressures (Fig. 3, *B*), which then drop to lower levels as the total blood volume falls to normal. If the ductus is large, the diastolic pressure remains permanently elevated, due again to a significant increase in peripheral resistance.

The following case report illustrates how very early the symptoms of cardiac disability may occur in the presence of a patent ductus.

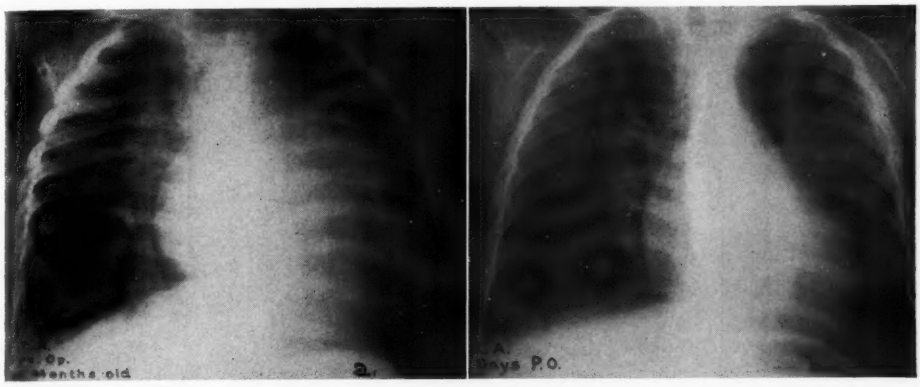

Fig. 4 (Case 1).—*a*, At 16 months of age the patient was in incipient cardiac failure due to patent ductus with marked dilatation of left heart. *b*, Twenty-six days after closure of the ductus, marked reduction in size of left heart had already occurred.

CASE REPORTS

CASE 1.—R. A., 16 months old, had had a heart murmur since he was 2 weeks of age. This was accompanied by attacks of dyspnea and a cough ascribed to recurrent pulmonary infection, but for which oxygen administration was necessary on several occasions. A roentgenogram taken at the age of 3 months showed cardiac enlargement. Thereafter, other films showed a progressive increase in heart size and prominence of the pulmonary vascular markings. At 15 months it was necessary to digitalize the patient in an effort to control heart failure. The murmur at this time was continuous and was heard maximally in the left second interspace. The blood pressure was 136/0 in the right arm; the electrocardiogram was normal. A diagnosis of patent ducturs arteriosus was made, and on Oct. 10, 1951, a ductus measuring 8 by 8 mm. was obliterated. In twenty-six days the heart returned to normal size (Fig. 4). The child had no further dyspnea and developed rapidly. Except for an inguinal hernia, he is normal in all respects.

Another patient with a history of mild cyanosis since birth died in cardiac failure at the age of 16 years without operation, a clinical diagnosis of interauricular septal defect having been made. At necropsy, a patent ductus was found as the only lesion, with unmistakable evidence that the flow of blood was and always had been, as in intrauterine life, from pulmonary artery to aorta (Fig. 5). This example of the association of cyanosis with patent ductus as the only anomaly is important since it is generally taught that it never occurs except in combination with other anomalies.[1, 21]

CASE 2.—J. J., aged 16 years was admitted to Stanford Hospital in acute cardiac failure of about five days' duration. At 9 months of age, following an attack of whooping cough,

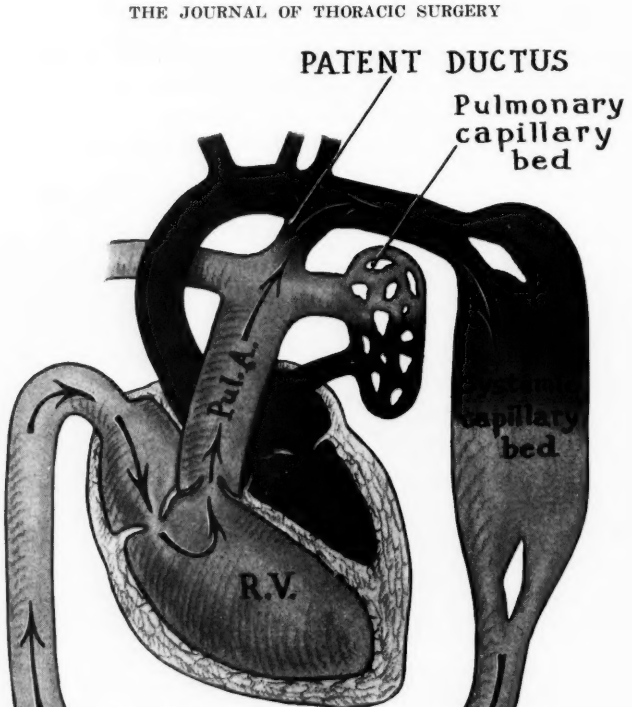

Fig. 5 (Case 2).—Schema of circulation when blood flows through ductus from pulmonary artery to aorta. The greater volume of blood flows through the right heart causing its dilatation. Hypertrophy also occurs because it is laboring against systemic resistance.

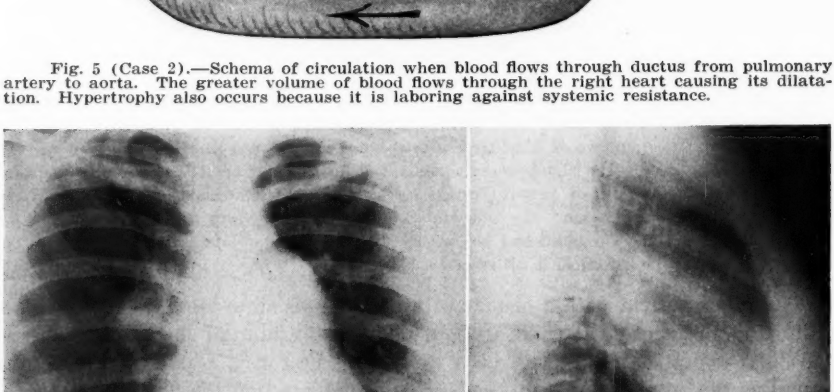

Fig. 6 (Case 2).—Roentgenograms disclosed a large right auricle and ventricle, enlarged pulmonary artery, and small left auricle and ventricle in presence of ductus transporting blood from pulmonary artery to aorta.

a cardiac murmur had been discovered. In recent years activity had become increasingly limited by fatigue and dyspnea. Although always a little flushed about the lips, she had been truly cyanotic only in the six months preceding admission, accompanied by increasing dyspnea and fatigue on exertion. A week before admission, vomiting occurred, which continued until she was placed in an oxygen tent. On physical examination, the child was well nourished, a little plump, with a distinct bluish color of lips and nails. She was dyspneic lying in bed.

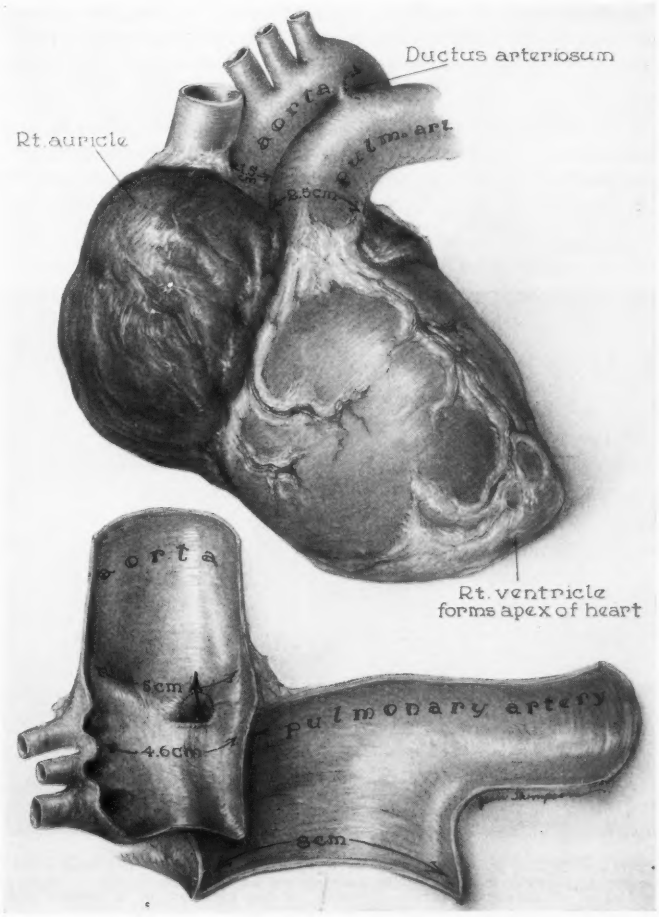

Fig. 7 (Case 2).—There is great dilatation of right auricle, and dilatation and hypertrophy of right ventricle which form the apex of the heart in presence of patent ductus transmitting blood from pulmonary artery to aorta. Note the excessive diameter and circumference of pulmonary artery as compared with aorta, and enlargement of aorta at site of entrance of ductus.

The chest and lungs were normal. The heart was slightly enlarged. There was a loud murmur maximal in the third interspace on the left, not well heard in the lung fields. It was thought to be systolic in timing. Blood pressure was 110/97, pulse rate 86.

Roentgenograms of the heart (Fig. 6) were interpreted as showing a very prominent and unusually large pulmonary artery, a bulging right auricle, a small left auricle, a small left ventricle, and small aorta. On fluoroscopy, a vigorous hilar dance was noted. The elec-

trocardiogram showed a marked right axis deviation, and abnormal P waves. The red cells numbered 6,100,000; hemoglobin 110 per cent or 18.7 Gm. per cent; the white cells numbered 8,450, 62 per cent polynuclears, 26 per cent lymphocytes, 8 per cent monocytes, 3 per cent basophiles. The urine had a specific gravity of 1.026 with 3 plus albumin and many pus cells. Cultures yielded *Escherichia Coli.*

Despite continuous oxygen administration the patient gradually became more cyanotic, dyspneic, and somnolent, and died thirteen days after admission. The clinical diagnosis was interauricular septal defect.

At necropsy, the heart weighed 400 grams. There was a remarkable hypertrophy of the right ventricle, and dilatation of the right auricle. The apex of the heart was formed by the right ventricle (Fig. 7). The left auricle and ventricle were small. The pulmonary artery was quite large, its circumference just beyond the valve being 8 cm.; at a corresponding point, the aorta was only 4.5 cm. in circumference. At the entrance of the patent ductus, the aorta measured 5 cm. in circumference. The tricuspid valve measured 11 cm. in circumference; the mitral valve 8 cm. in circumference. The right ventricle when dissected out weighed 167 Gm.; the left ventricle weighed only 78 Gm. Microscopically the fibers from the right ventricle were slightly larger than those of the left, and the nuclei were generally larger and more blunt.

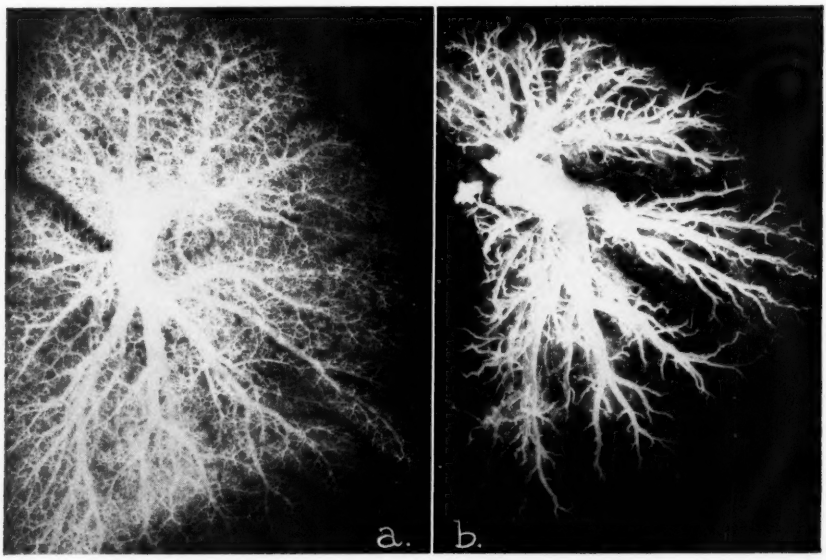

Fig. 8 (Case 2).—*a*, Roentgenogram of normal lung following injection with lead carbonate and mercuric sulfide in gelatin. *b*, Roentgenogram of lung (Case 2) similarly injected revealed evidence of pulmonary arteriosclerosis in the presence of a patent ductus through which blood flowed from pulmonary artery to aorta.

The pulmonary arterial tree was injected with lead carbonate, plus mercuric sulfide suspended in gelatin. When compared with a normal lung similarly injected (Fig. 8), it showed a truncated, blunted arterial tree, suggesting long-standing pulmonary hypertension and arteriosclerosis. Microscopically many of the small arteries and arterioles had thickened walls and narrowed lumina, and most of these had thickened intima with reduplication of the elastic laminas. A cross section of the aorta, patent ductus, and pulmonary artery showed the wall of the pulmonary artery definitely thicker than the aortic wall, and except for a definite heavy elastica interna, it was similar in structure.

A very exacting search revealed no lesion other than the patent ductus arteriosus which in the relaxed state measured 0.5 cm. in diameter but could easily be distended to 1 cm. in diameter. All evidence indicated that for her entire lifetime, this patient's ductus had been transporting blood from the pulmonary artery into the aorta,[4] thus providing a larger volume flow of blood through the right heart and systemic circulation than through the left heart and pulmonary circulation. The expected response to this increased volume flow was: (1) a preponderant dilatation of the right heart because of the larger volume of blood flowing through it; (2) a preponderant hypertrophy of the right heart due to its being in direct communication with the peripheral resistance of the systemic circulation against which it had labored for a lifetime; (3) a marked right axis deviation; (4) a pulmonary artery whose lumen was larger than that of the aorta with a wall thicker than the aortic wall; (5) an aortic lumen which was 4.5 cm. in circumference at its origin and 5 cm. at the entrance of the ductus; (6) a definite cyanosis in life, indicating an admixture of venous blood to the systemic blood by way of the ductus; (7) a high diastolic pressure since blood flowed through the ductus into the systemic circulation during diastole rather than out of it.

Summed up, these deviations provide unequivocal evidence that the flow of blood through the ductus was in this instance from pulmonary artery into the aorta. Seldom if ever has this type of ductus been recognized by the surgeon at the operating table, probably because this rarely encountered condition is usually mistakenly diagnosed, and surgery is not considered advisable. This may have been fortunate, as it seems highly probable that closure of the ductus in this 16-year-old girl with such a preponderantly developed right heart would have been incompatible with life. Recognition of this condition at an earlier age might permit successful surgical correction.

A second equally serious threat which may occur at an early age is a bacterial endocarditis and endarteritis involving the ductus itself, the adjacent pulmonary artery, or the aorta.[14] In the eighty cases of patent ductus studied by Bullock and his associates,[4] fatal bacterial endocarditis occurred in 53 per cent. Four of our cases were complicated by an endarteritis and a bacterial septicemia, one when the patient was 7, one at 10, and two at 11 years of age.

CASE 3.—I. R., a thin underdeveloped girl, was first seen at the age of 7, in November, 1944, for a cardiac murmur since birth. A diagnosis of patent ductus and megacolon was made. Operation on the ductus was not advised by the pediatricians because "the child had had no symptoms." Exactly one month later she was readmitted to the hospital acutely ill with high fever, marked anemia, and subacute bacterial endarteritis, for which she received over 12,000,000 units of penicillin in the course of three months' hospital care with subsidence of all symptoms.

She re-entered the hospital in January, 1946, perfectly well, but operation was now considered advisable as a prophylaxis against future trouble. Closure of a large ductus, 1 cm. in diameter, by multiple ligations was followed by a gain of fourteen pounds in a year's time, and by a marked reduction in the size of the heart (Fig. 9) comparable with the reduction in cardiac size that follows excision of a peripheral arteriovenous fistula. A blood pressure of 105/60 rose immediately after operation to 134/96. The following day a blood pressure of 138/110 was found, a week later 120/82, and two years later 115/80.

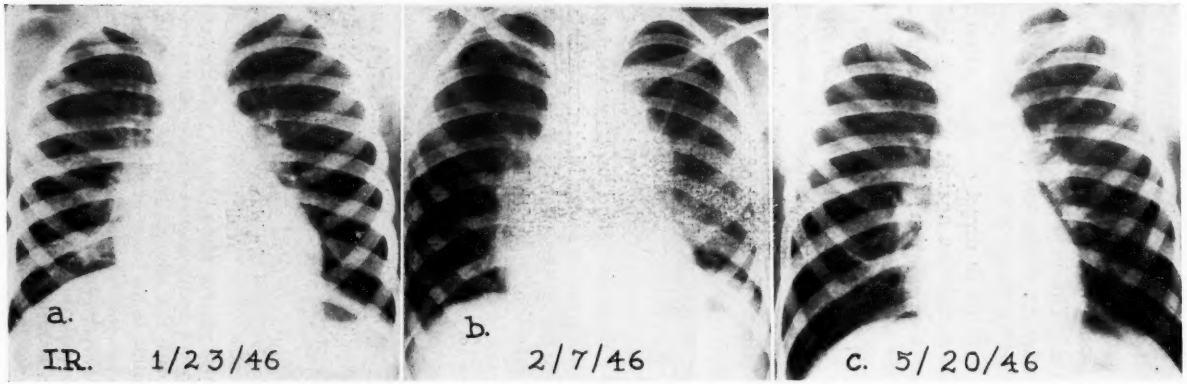

Fig. 9 (Case 3).—*a*, Large heart in presence of a patent ductus complicated by bacterial endarteritis. *b*, immediately following ligation of ductus. *c*, Three months later there is marked reduction in size of heart comparable with the reduction of cardiac size that follows elimination of a peripheral fistula.

Fig. 10 (Case 4).—Roentgenograms disclosed *a*, dilatation of heart in presence of a recurrent ductus, ligated seven years previously; *b*, biloculate aneurysm of ductus at site of triple religation, ductus again patent; *c*, following division of ductus, the aneurysm is no longer seen, and a bacteriemia previously resistant to all antibiotic therapy was cured.

In two cases of open ductus complicated by bacterial endarteritis and septicemia, aneurysmal dilatations occurred, one of the ductus itself and one of the pulmonary artery.

CASE 4.—P. P., aged 11 years, was found to have a patent ductus at the age of 9 months. In 1943, at 4 years of age, because of shortness of breath and an enlarging heart, the ductus was closed at another hospital with a single tape ligature. Within one month after the ligation, the murmur and symptoms had recurred. In August, 1950, at the age of 11, she entered Stanford Hospital for religation. The only complaint was shortness of breath on exertion. Upon physical examination, she appeared to be of normal weight and development for her age. There was moderate cardiac enlargement (Fig. 10), and a loud continuous murmur could be heard to the left of the sternum at the second interspace. The blood pressure was 112/54, the pulse rate 92. On Aug. 24, 1950, the ductus was exposed through the old incision in the third intercostal space anteriorly. There was dense scar tissue surrounding the ductus which was isolated with considerable difficulty, and a single ligature was passed around it for subsequent ligation. In attempting to free the displaced adherent vagus nerve from the scarred anterior surface to permit the application of a second ligature, the ductus was suddenly entered with profuse hemorrhage. It was evident that the nerve had formed part of the wall of the ductus. While bleeding was being controlled by finger pressure, a Potts serrated clamp was applied to the aortic arch proximal to the ductus, and the accidental rent was closed by several interrupted silk sutures. Three braided silk ligatures were then applied to tie off the ductus, division being thought inadvisable. On discharge from the hospital, August 31, a blood pressure of 130/100 and a pulse rate of 88 were recorded. Six weeks later chills and fever developed, and the murmur to the left of the sternum reappeared, at first of mild intensity but becoming increasingly loud under observation. The blood pressure was 104/60, pulse rate 120. A blood culture yielded a *staphylococcus albus,* which was resistant to penicillin, streptomycin, Aureomycin, terramycin, and to some extent Chloromycetin. The patient was treated with chloramphenicol and the cultures became sterile for a few days only, when the organisms reappeared. During this period she was febrile, obviously ill, had repeated epistaxis requiring nose packing, and looked drawn and pale. Nearly all her hair fell out. A course of bacitracin and penicillin was given without effect upon the blood stream infection.

On Jan. 11, 1951, the ductus area was again exposed through a posterolateral incision in the fourth interspace. The dissection at this time was tedious and time consuming, the entire operation lasting nine hours. After considerable difficulty, a bilobed aneurysmal swelling involving the anterior wall of the ductus was partially isolated (Fig. 11). Arterial blood could be seen coursing through it with each systole. The arch of the aorta, the left subclavian artery, and the pulmonary artery distal to the aneurysm were completely mobilized. The pericardium was opened, and a Crafoord clamp was applied to the left main pulmonary artery within the pericardium. Another clamp of the same type was applied to the distal pulmonary artery. A large side-occluding clamp of the Potts type was applied to the aorta. The ductus and the aneurysm having been completely isolated from the systemic and pulmonic circulations, the ductus was divided at the site of the aneurysmal dilatation. Two braided silk sutures were found lying within the lumen, embedded in vegetations on the ductus wall, and surrounded by granulation tissue. These foreign bodies undoubtedly accounted for the stubborn persistence of the circulating organisms. The aneurysmal wall and surrounding scar were resected; the openings into the aorta and pulmonary artery were closed with arterial silk, it being necessary to reconstruct partially the pulmonary artery. Adjacent scar tissue was used to reinforce the suture lines. Cultures taken from the vegetations grew the same organisms as had been present in the blood. All postoperative blood cultures were negative, and the patient made an uneventful recovery. A blood pressure of 112/88 was recorded on the evening of operation and has remained normal since that time. The patient has regained normal strength and health and has returned to school.

Of equal interest was the occurrence of an aneurysm of the pulmonary artery[15, 16] located exactly opposite the entrance of the ductus. This aneurysm was also the result of an endarteritis, and a stubborn blood stream infection.

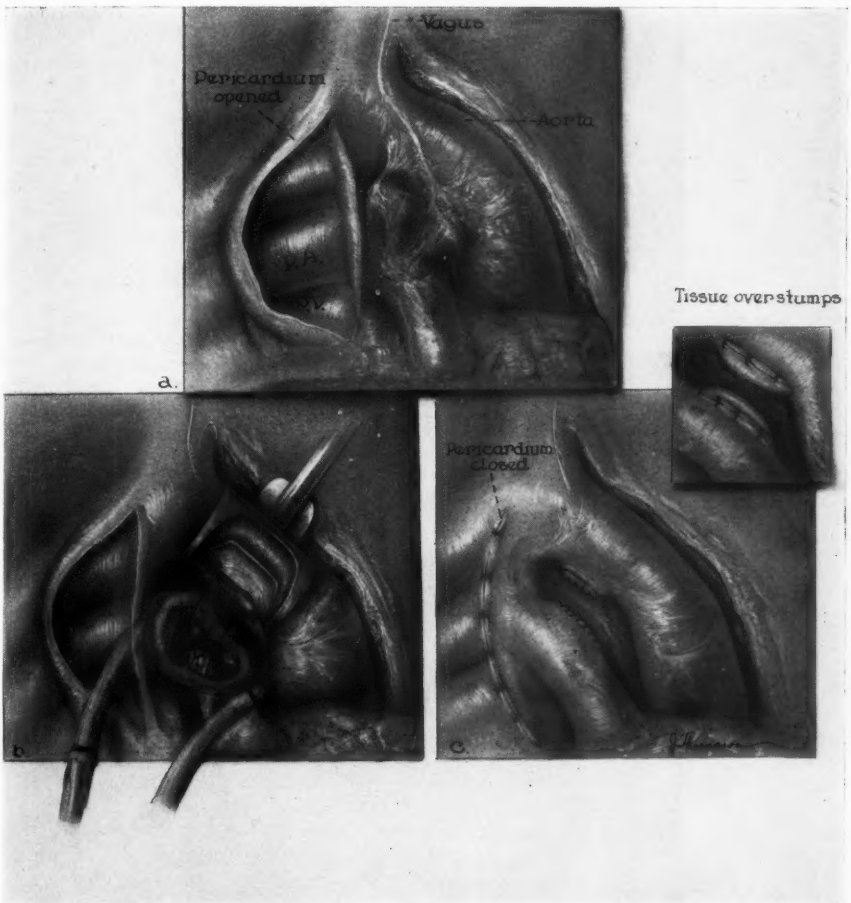

Fig. 11 (Case 4).—*a*, Biloculate aneurysm of patent ductus which followed triple ligation of ductus, and a blood stream infection with *Staphylococcus albus* which failed to yield to any antibiotics. *b*, Following complete isolation of the aneurysm the ductus was divided at the site of aneurysmal dilatation. Two silk ligatures found imbedded in vegetations were removed. *c*, Opening in the aorta was closed by a continuous suture, pulmonary artery reconstructed from ductal wall. Following operation blood stream was sterile.

CASE 5.—L. P., aged 14 years, entered the hospital with a diagnosis of congenital heart disease. A murmur had been present since birth. During her early childhood she was normal in all respects, except that she tired easily. Three years before entry she began having fainting spells when she was tired or nervous. About this time also she had what was called influenza, which was followed by a persistent fever which was found to be due to a blood stream infection. She entered a hospital where she remained for five

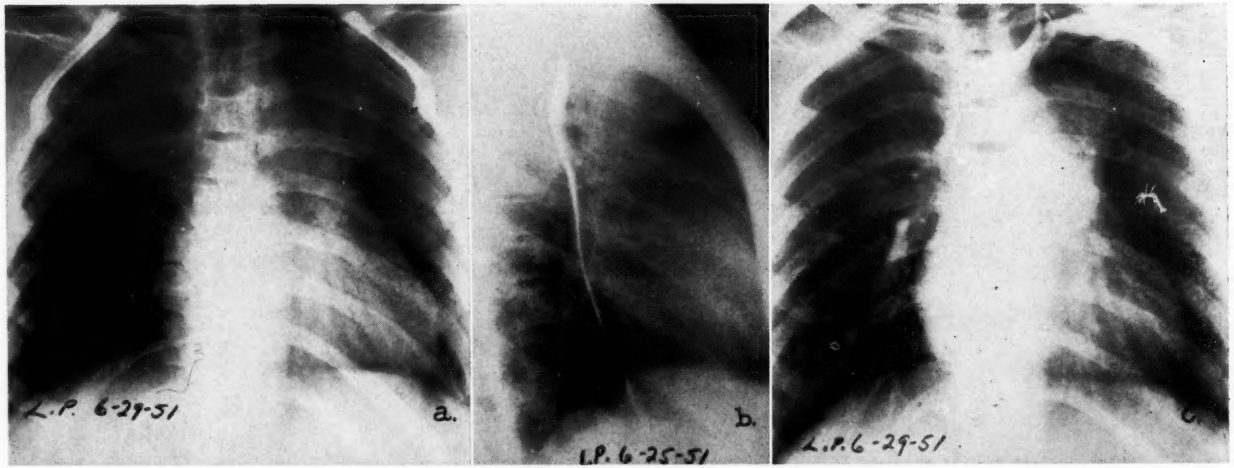

Fig. 12 (Case 5).—*a* and *b*, Roentgenograms revealed aneurysm of pulmonary artery. *c*, Angiogram revealed the aneurysm only partly opacified indicating the presence of a thrombus.

months and received 992 injections of penicillin! Following this illness she was fairly well. Upon examination she was a normally developed girl whose abnormal physical findings were limited to the heart. The blood pressure was 120/60. The heart was slightly enlarged. There was a continuous murmur to the left of the sternum in the second interspace. Roentgenograms (Fig. 12) revealed an aneurysmal swelling either of the left pulmonary artery or of the ductus. Cardiac catheterization was performed, with the findings as shown in Table I.

TABLE I

SITE OF CATHETER		OXYGEN ANALYSIS VOLUMES PER CENT
Superior vena cava		13.3
Right ventricle		13.5
Pulmonary artery		15.4
Femoral artery		17.4
PRESSURE MEASUREMENTS		
Pulmonary artery	48/18	
Right ventricle	50/6	
Femoral artery	113/65	

Angiocardiograms (Fig. 12) revealed partial opacification of the aneurysm and reopacification of the pulmonary vessels, indicating a left to right shunt. On July 7, 1951, the ductus was exposed through a posterolateral incision in the fifth interspace (Fig. 13, *a*). The ductus measured 1 by 0.7 cm. The pulmonary artery opposite the entry of the ductus was greatly dilated, forming an aneurysm which measured 4 cm. in diameter. The aneurysm contained a thrombus as was suggested by the roentgenograms. The ductus was

Fig. 13 (Case 5).—*a*, Large 4 cm. sized aneurysm of pulmonary artery opposite opening of patent ductus. *b*, Ductus closed by multiple ligations; aneurysm surrounded by irritating type of Cellophane. Patient is well.

obliterated by the ligature-transfixion method, and the aneurysm itself was wrapped with two layers of cellophane (Fig. 13, *b*), the inner layer being of the irritating type, and the outer, the nonreacting type. Her postoperative recovery was uneventful. On August 28, a blood pressure of 110/80 and a pulse rate of 78 were recorded. She now attends school

and participates in moderate athletics, but still has fainting spells occasionally. Roentgenograms reveal no change in the size of the aneurysm.

A pair of identical twins (Fig. 14) presented almost identical clinical pictures with this important difference: at operation one ductus measured one millimeter greater in diameter and this twin showed greater impairment of circulation as judged by a greater dilatation of the heart, and a lower diastolic pressure.

CASE 6.—Marjorie J., aged 3½ years, had a birth weight of 2 pounds, 13 ounces and was an incubator baby for six weeks. Her development was slow; she walked at 18 months and thereafter fatigued easily in play. A murmur was first noted at 2 weeks of age but there was no cyanosis. She was admitted for ligation of ductus on Dec. 19, 1950. On physical examination the heart was large (Fig. 15, *b*), a loud machinery-like continuous murmur was transmitted into neck and back. The pulse rate was 94, blood pressure in left arm, 102/0, right leg, 132/20; red blood cells 4,800,000, hemoglobin 14.5 Gm. per cent, white cells 9,600, 42 per cent neutrophils, 46 per cent lymphocytes, 2 per cent eosinophils, 2 per cent basophils, 8 per cent monocytes. At operation, the ductus was 18 mm. long and 10 mm. in diameter at the aortic end, 8 mm. at the pulmonic end. The blood pressure was elevated immediately following operation (Fig. 16, *A*), and the patient has remained well.

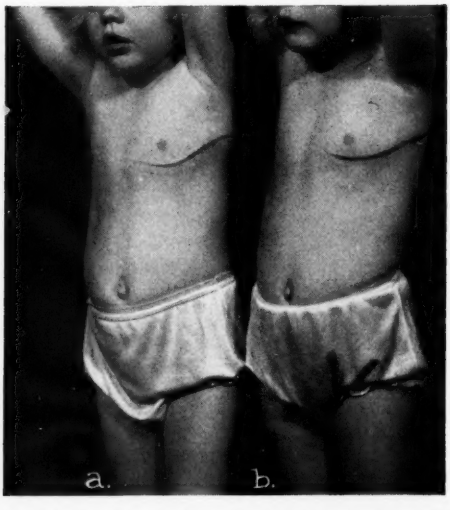

Fig. 14 (Cases 6 and 7).—Identical twins with identical patent ductus who showed almost identical clinical pictures, with identical responses following operation (see Figs. 15 and 16).

CASE 7.—Mary J., age 3½ years, had a birth weight of 2 pounds, 14 ounces and was an incubator baby for six weeks; she walked at 17 months. She was admitted for ligation of the ductus on Dec. 19, 1950. The heart was enlarged (Fig. 15, *a*), but less so than that of her twin sister. A loud machinery-like continuous murmur was heard best in the second interspace on the left but was transmitted over the whole precordium; the pulse rate was 110, blood pressure in the left arm, 90/40; in the left leg, 104/46; red blood cells numbered 5,300,000 hemoglobin 14.5 Gm. per cent, white cells 5,600, 40 per cent neutrophils, 2 per cent eosinophils, 1 per cent basophils, 50 per cent lymphocytes, 7 per cent monocytes. At operation, the ductus appeared exactly similar to that of her twin though slightly smaller; it was 18 mm. long, 9 mm. in diameter at the aortic end, and 7 mm. at the pulmonic end. The blood pressure was elevated abnormally immediately after closure of the ductus but fell to 110/80 on discharge (Fig. 16, *B*). She has remained well.

Religation of a Recurrent Ductus: Religation[11] was necessary in our very first patient operated upon in December, 1939, when the ductus was closed with a single ligature, that being the procedure then in vogue. At reoperation, the ductus was found as large as at the first operation, with only small masses of scar tissue present to disclose the site of the previous ligation. The ductus was isolated without difficulty and tied at both aortic and pulmonic ends with a 3 mm. wide tape. In view of the almost normal appearance of the ductus at this

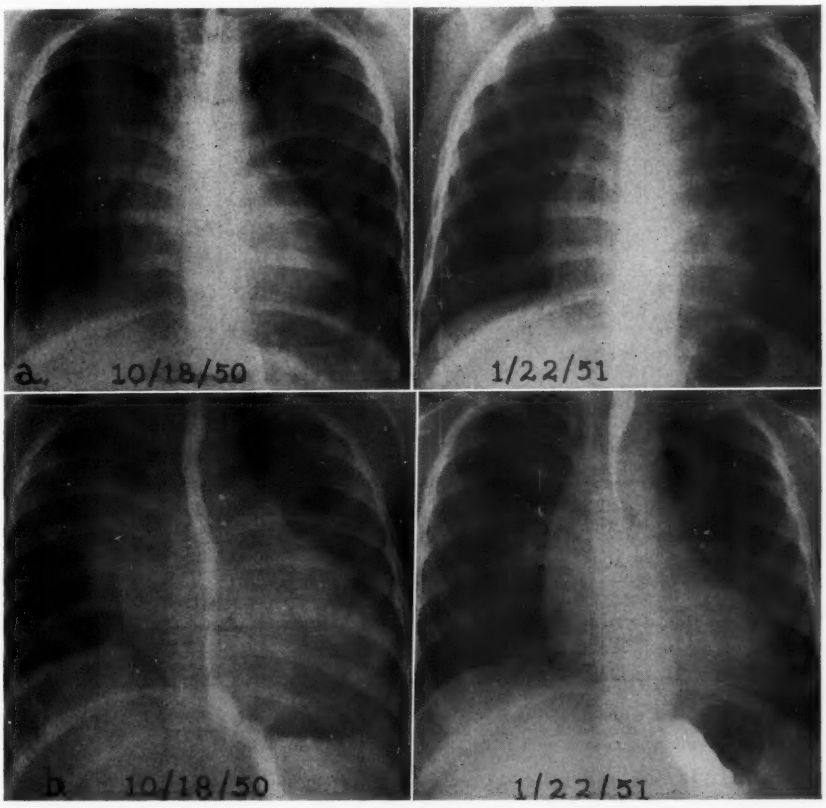

Fig. 15 (Cases 6 and 7).—Roentgenograms of chests of identical twins, *a* and *b*, before and after triple ligation of patent ductus on Dec. 19, 1950, showing decrease in size of heart mainly due to a smaller left ventricle. Note decrease also in hilar markings. In twin *b* whose heart was definitely more dilated, the ductus measured 1 mm. larger in diameter.

second operation, our medical colleagues questioned whether the ductus had actually been ligated at the first operation. Unequivocal evidence that the ductus had been properly occluded was available from the operative records. At the operating table, the blood pressure rose immediately after this first ligation from 110/70 to 130/100 and the pulse rate dropped from 100 to 70, a phenomenon directly attributable to the increased blood volume in the presence of a ductus and to the increased peripheral resistance imposed by closure of the

ductus. On December 19, ten days after ligation, the blood pressure was 120/90 as compared with a preoperative reading of 130/70. On May 20, 1940, when the murmur was again heard in full force indicating reopening of the ductus, the blood pressure was 124/60. At the second operation on April 5, 1941, closure of the ductus was followed immediately by a rise in pressure from

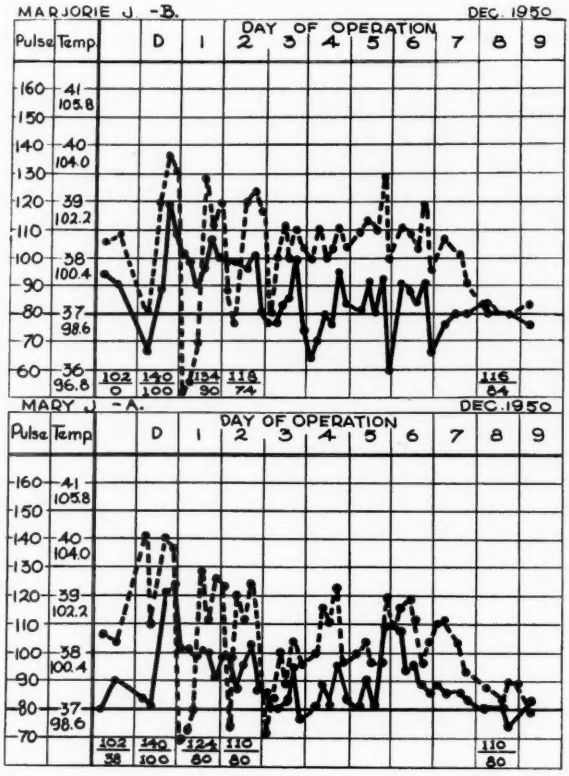

Fig. 16 (Cases 6 and 7).—Temperature and pulse chart of twins, *a* and *b*, showing almost identical response to closure of ductus. Note increase in blood pressure from 102/0 to 140/100 on day of operation in twin *b*, and from 102/38 to 140/100 in twin *a*.

128/50 to 148/106 and by a decrease in pulse rate from 116 to 100. On discharge from the hospital, the pressure was 112/84, and this normal pressure has been maintained. The patient has married and has had several children.

At the operating table, this evidence of an increase in blood pressure on trial closure of the ductus by digital pressure is reassuring evidence, first, that the proper structure has been isolated and, second, that its permanent closure can be tolerated with impunity without depriving the patient of a shunt necessary for proper aeration of the blood as might occur in connection with a patent ductus accompanying a mild, unrecognized tetralogy of Fallot.

The fortuitous passing of a catheter from pulmonary artery through the ductus into the aorta is worthy of mention to illustrate the effect of a ductus upon oxygen values at various sites.

CASE 8.—L. P., was 6 years old. The mother had had rubella at one month of gestation. Murmur was first noted in the infant at 10 weeks of age. At 2½ years, a thoracic deformity (scoliosis) was first noted. The patient was admitted to the hospital in September, 1949, for cardiac catheterization studies, there being a question of an interauricular

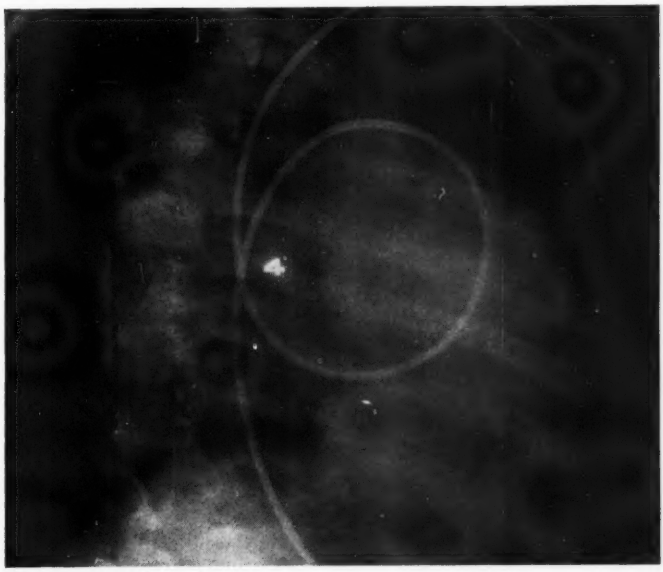

Fig. 17 (Case 8).—Roentgenogram revealed catheter looped through patent ductus into the aorta.

septal defect. A cardiophonogram recorded a prominent continuous murmur in the pulmonic area, and a systolic and diastolic murmur at the apex, which was thought to be the same murmur present in the pulmonic area. The roentgenogram of the chest was interpreted as showing both right and left ventricular enlargement and marked enlargement of the pulmonary vascular bed.

Cardiac catheterization resulted in threading the catheter from the pulmonary artery into the aorta (Fig. 17).

TABLE II

SITE OF CATHETER	OXYGEN VOLUMES PER CENT	PRESSURE MEASUREMENTS
Superior vena cava	10.9	
Right auricle	10.8	5.5 mm. Hg
Right ventricle	10.5	61/11
Pulmonary artery	13.4	60/37
Aorta	13.4	84/45
Oxygen capacity	15.0	
Oxygen saturation	89%	

On Nov. 9, 1949, the patent ductus was closed by multiple ligations. The ductus measured 1.4 cm. in diameter. Before ligation of the ductus, the pulmonary conus had a diameter of 2.3 cm., after ligation, 1.5 cm. The blood pressure rose to 120/80 immediately after ligation and the following day was 130/110, but subsequently dropped again to 104/80.

Pregnancy as a complication of a patent ductus is illustrated by the following patient:

CASE 9.—Mrs. O. M. N., 26 years of age, was known to have had a heart murmur since the age of 8 years. Although she had been followed by a competent pediatrician before that time, no murmur had been noted, and after its discovery it apparently was rather evanescent. She played basketball in high school without discomfort. Since an attack of measles one year before admission, there had been some shortness of breath, which had become very distressing since becoming pregnant about a month before admission. Formerly a choir singer, she could no longer sing due to dyspnea. The least effort at work tired and distressed her. Her heart "pounded" on excitement. There had been no cyanosis, no fever, and no swelling of the ankles.

Physical examination disclosed a machinery-like continuous murmur over the second interspace to the left of the sternum, the systolic phase audible over the whole precordium; blood pressure 140/70, pulse rate 120, after exercising, 148. The electrocardiogram was normal; red blood cells numbered 3,500,000, hemoglobin 80 or 13 Gm. per 100 c.c., white cells 5,100. On March 18, 1948, the ductus was divided and the two cut ends were closed with continuous sutures. On closing the ductus, the pressure rose from 110/70 to 110/90. Recovery was uneventful and she left the hospital on March 28, only to lose the 1-month-old fetus three days after returning home. Blood pressure one month later was 136/84.

The case is cited to illustrate the baneful effect of pregnancy on a heart already burdened by a patent ductus.[17] The increased total blood volume[18] and increased cardiac output which occur in pregnancy together with the increased output and increased blood volume due to the ductus both combined to increase the cardiac burden and the dyspnea that developed during the pregnancy. The diminished cardiac reserve imposed by a ductus in adult years as observed in this pregnant woman provides additional justification for advising closure of the ductus in childhood.

Five patients were explored by thoracotomy for symptoms and signs indistinguishable from those of an open ductus, but no ductus was found.

CASE 10.—V. M., 8 years of age, had a cardiac murmur discovered at the age of 26 months. She had been on restricted activity, but had few, if any, cardiac symptoms. Upon physical examination, there was a harsh systolic murmur over the precordium, loudest over the second and third interspace to the left of the sternum. A thrill was present in this region. Posteriorly, the murmur could be heard but it was less loud. The blood pressure was 112/72. An electrocardiogram showed right axis deviation. Roentgenograms revealed a greatly enlarged pulmonary conus. This was confirmed by angiocardiograms which were interpreted as showing reopacification of the pulmonary artery from the aorta, suggesting a patent ductus. On April 19, 1941, the left thorax was explored through an anterior incision. A greatly enlarged pulmonary artery was found, and at its origin there was a marked thrill and a loud bruit heard through a sterile stethoscope. Pressure could not completely obliterate it, although it could be reduced in intensity. No ductus was found. A diagnosis of aortopulmonary artery septal defect was made, closure of which was not considered feasible because of its location. She was last seen in 1945 when she was having growing pains suggesting the possibility of rheumatic fever.

CASE 11.—M. S., a 19-year-old Japanese girl, was known to have had heart trouble for five years. There had been no symptoms until one year before entry, when palpitation and exertional dyspnea developed. Upon examination, there was a very loud, continuous murmur and thrill in the third left interspace, heard loudest approximately 6 cm. from the midline. The murmur could also be heard over the whole left thorax, and was quite loud in the back. Roentgenograms showed a large heart suggestive of patent ductus. The blood pressure was 108/40. At operation, on Jan. 15, 1949, an obliterated ligamentum arteriosum was found. A marked thrill was present at the base of the pulmonary artery, just at its point of origin. Pressure in this region obliterated the thrill. It was not considered technically feasible to close the defect due to its close proximity to the heart. There has been no progression of symptoms since the operation.

CASE 12.—J. A. W., aged 6 years, was first suspected of having cardiac trouble at the age of 6 because of a persistent tachycardia and systolic murmur. Frequent colds, easy fatigability, and a bluish pallor have been noted frequently since.

Physical examination disclosed an enlarged heart, a prominent pulmonary artery, a harsh systolic murmur along the left sternal margin, and a soft diastolic murmur in the midsternal area. A blood pressure of 100/82, a pulse rate of 90, and left axis deviation were recorded.

Catheterization studies suggested an atypical interventricular septal defect but a patent ductus could not be ruled out (Table III).

TABLE III

SITE OF CATHETER	OXYGEN CONTENT VOLUMES PER CENT	PRESSURE MEASUREMENTS
Inferior vena cava	11.0	
Superior vena cava	13	
Right auricle	13.6	
Right ventricle	16.2	84/7 mm. Hg
Pulmonary artery	15.9	84/44 mm. Hg
Femoral artery	16.9	86/54 mm. Hg
Femoral artery	92% saturated	
Hemoglobin	12.8 grams	

At operation on June 22, 1950, there was no ductus present, the heart was large; the left pulmonary artery was greatly dilated with a diameter of 2.5 cm., whereas the arch of the aorta seemed hypoplastic with a diameter of only 1.5 cm. Only a mild systolic thrill was present over the pulmonary artery. A high interventricular septal defect was thought to be most probable.

CASE 13.—M. S., 25 years of age, had always been short of breath, easily fatigued, and pale since birth. Married seven years previously, there had been three pregnancies with two living children. Six months before admission to hospital, while five months pregnant, she had become more dyspneic, unable to do her work, and fainted on several occasions. In the two months before admission there had been fever, with chills, severe sweating, severe headaches, and a blood culture had yielded an organism identified later as an anaerobic, gram-positive member of the Actinomycetales group for which 200,000 units of penicillin had been administered every three hours for a week.

Physical examination showed marked pallor, dyspnea while lying in bed, visible thrust of the precordium, palpable thrill over the whole precordium, and a loud machinery-like murmur, most intense over the left third interspace, but heard over the whole precordium, in the left axilla and back and in the neck. The temperature was 38° C., the pulse rate 110, respiration 20, blood pressure 142/58; red blood cells 4.9 million, hemoglobin 75 per cent, venous pressure 10 cm. water; circulation time, arm to tongue, 34 seconds.

The clinical diagnosis by all who saw her was patent ductus with bacterial endarteritis. Under observation and with penicillin therapy, the temperature improved although it remained somewhat elevated, and the blood pressure became 160/0, suggesting an increasing shunt; accordingly early operation was advised.

At operation on Oct. 1, 1947, no ductus was found. The thrill which was greatest immediately over the origin of the pulmonary artery could be obliterated only by almost complete closure of the pulmonary artery by pressure. A high fever and rapid pulse followed the operation; the patient lost ground rapidly and died in cardiac failure six days after operation.

At necropsy, the foramen ovale measured 1.5 by 0.5 cm. with vegetations on its margin. There was an interventricular septal defect (Fig. 18) measuring 1.7 by 1.2 cm., and an aneurysm of the sinus of Valsalva providing a communication into the right ventricle, measuring 0.2 by 0.3 cm. The pulmonary artery was greatly dilated, measuring 9.8 cm. in circumference, the aorta, 6.2 cm. The tricuspid valve measured 14.7 cm. in circumference, the mitral valve, 11.7 cm. Endocarditis involved the aneurysm, the septal defects, and the pulmonary artery.

CASE 14.—A. A., 33 years of age, entered the hospital with the complaint of increasing dyspnea. There had been a heart murmur and attacks of cyanosis in infancy, but no

serious symptoms until ten years previously when an abrupt attack of palpitation occurred lasting about four hours. Examination then showed a dextrocardia, and a systolic murmur over the right precordium. During the two years before entry, he had noticed a gradually increasing fatigability and dyspnea. One year before entry he had hemoptysis and an attack of vertigo. Upon examination, the blood pressure was 120/70. The neck veins pulsated. The heart was on the right. The murmur was now clearly continuous in the right second interspace, with a rough, loud systolic component at the third and fourth right interspace. Roentgenograms showed an enlarged left ventricle and congested pulmonary vessels which pulsated actively. Electrocardiograms showed abnormal T waves and abnormal precordial leads. Laboratory and blood studies were normal. Cardiac catheterization* revealed an oxygen content of 15.7 volumes per cent in the right ventricle and 20.8 in the pulmonary artery. The femoral artery blood was 96 per cent saturated. The right ventricular pressure was 77/0, and the pulmonary artery 20/-2. These findings were considered as being compatible with patent ductus arteriosus and pulmonic stenosis. Operation was performed on April 1, 1952, through a right posterolateral incision. The

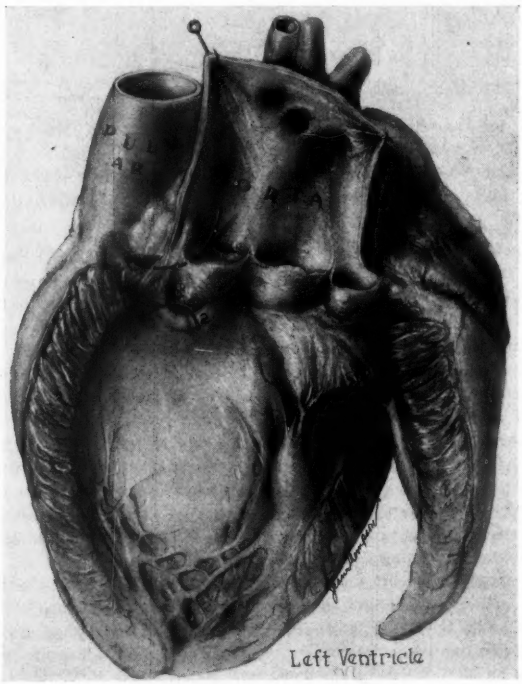

Fig. 18 (Case 13).—Following thoracotomy for presumed ductus which was not found, the patient died of bacterial endocarditis. Two lesions accounted for signs and symptoms indistinguishable from those of ductus, namely, (1) an aneurysm of the sinus of Valsalva transmitting blood from aorta into pulmonary artery, and (2) a high interventricular sepal defect.

ductus was not patent. After opening the pericardium, it was found that the maximal thrill was in the myocardium at the beginning of the aorta and pulmonary artery, giving the impression of a defect near or perhaps just distal to the valves. It was impossible to stop the thrill by pressure. Due to the proximity of the coronary vessels, it was felt inadvisable to attempt closure of the defect. The postoperative recovery was uneventful.

*Performed by Dr. Hultgren.

Operative Technique.—The following important details characterize the operation as performed at the present time.

1. Operative approach either through an anterior incision in the third interspace with division of the third and second costal cartilages, or through a posterolateral incision in the fourth interspace with separation of the ribs but without either division or excision of a rib. The former incision is preferred in boys, and the latter in girls or in young women sensitive to the appearance of a scar.

2. Identification of the site of the ductus by palpating for the area of greatest thrill.

3. Identification of the vagus nerve and of the recurrent laryngeal nerve as it runs underneath the ductus.

4. Isolation of the transverse arch proximal to the left subclavian artery for the subsequent application of a Potts serrated clamp or an occluding clamp such as the cloth-shod Crafoord clamp just prior to ligation of the ductus.

5. Isolation of the anterior, lateral, and medial walls of the ductus close to the thicker walled aortic end rather than near the pulmonic end. The transverse diameter of the ductus is measured, and this measurement is used in locating accurately the site of the posterior wall of the ductus so as to stay well away from it and thus avoid injuring it during dissection of the posterior periductal tissues. Once the tip of the right-angled clamp has penetrated the periductal tissues to reappear on the side opposite the operator, the complete isolation of the ductus is a matter of careful division and separation of tissues now easily observed and identified. The full length of the ductus having been properly developed, the effect of its closure on blood pressure and pulse is determined and measurements are taken. The decision is then made whether to apply multiple ligatures in continuity[19] or to apply four clamps, divide between the central two clamps, remove these two clamps, and suture the open cuff projecting beyond the remaining two clamps.[20] Recently, a decision to divide and suture the ductus in a 4-year-old child was made because of its large size, 1.5 cm. in diameter. To apply a ligature to such a large ductus invites the real danger of fracturing the wall of the ductus while attempting to drive home the knot. Division and suture were easily performed.

A word of caution when applying multiple ligatures is in order. In applying the first ligature to the aortic end of the ductus, the ligature is anchored in place by taking a few bites of the periductal and periaortic tissues so that in tying, the ligature will not slip from the end to the center of the ductus. Also before tying this ligature, a serrated Potts clamp[21] or a Crafoord clamp is applied to the transverse arch in the area previously mobilized to be released again as soon as the ligature has been tied. This abolishes pulsation in the ductus during the act of ligation, thus reducing the hazard of fracturing the wall of the ductus. The re-establishment of an open ductus following ligation may well have its origin in injury to its wall at the moment of tying a knot against the resistance of a vigorously pulsating blood stream. A ligature is next applied to the pulmonic end of the ductus, it also being anchored in place by taking a few bites of the periductal tissues just where it joins the pulmonary artery.

TABLE IV. AGE AT OPERATION

AGE (YR.)	NO.	AGE (YR.)	NO.
0 to 2	5	16 to 20	6
3 to 5	25	21 to 25	3
6 to 10	23	26 to 30	2
11 to 15	10	31 to 35	1

Two transfixion ligatures are then applied to the ductus in the segment between the two ligatures previously placed. The site of ligation is bathed in penicillin solution, the pleura overlying the ductus is closed, and the chest wound resutured. Intercostal drainage is rarely used. Very little blood is administered by transfusion unless real blood loss has occurred since closure of the ductus invariably causes a temporary rise in blood pressure due to the autotransfusion that occurs at the moment of ligation, attributable to the increased blood volume that has occurred in the presence of the ductus.

TABLE V. REASONS FOR OPERATING

Clinical diagnosis with few or no symptoms	30
Clinical diagnosis with enlarged or failing heart	5
Clinical diagnosis with moderate to severe dyspnea and fatigability, poor growth, or endocarditis	40

Another word of caution is in order. If, at any time, bleeding should occur during isolation of the posterior wall of the ductus and this bleeding is easily controlled by mild digital pressure, further dissection is immediately discontinued. Two courses of action are then available. If no further bleeding occurs upon release of digital pressure, several minutes later, the operation may be terminated and the wound closed. After the elapse of six months to one year, reoperation may again be considered and performed without difficulty. This procedure was employed in one case in which this mild bleeding occurred during isolation of the posterior wall of the ductus. Disaster might have followed if the operation had been pursued further. Six months later complete success marked the second operation and the child is now well. The only death occurred in a young 7-year-old patient in whom this precaution was not observed when slight, easily controlled bleeding occurred while isolating the posterior wall of the ductus. In pursuing the dissection further, excessive bleeding was encountered, requiring transfusion of 2 liters of alien blood (presumably properly matched) and 2 liters of her own blood recovered at the operating table. De-

TABLE VI. ASSOCIATED NONCARDIAC ANOMALIES

Cataract and strabismus*	1
Cataract*	1
Cataract and clubfoot	1
Cataracts, bilateral, internal strabismus, thoracic deformity, displaced lenses*	1
Cataracts, nystagmus†	1
Strabismus	1
Undescended testes	1
Hypogonadism	1
Megacolon	1
Thoracic deformity*	1
Harelip, cleft palate, syndactylism	1

*Mother had rubella at about one month of gestation.
†Mother had rubella at two to three months of gestation.

spite the successful division of the ductus, complete anuria followed and she died in convulsions seventy-two hours after operation.

A second course of action would be to continue the operation by isolating all structures proximal and distal to the ductus, as described in Case 4, and to proceed with division of the ductus. This decision may, in some measure, depend upon the experience of the operator.

Tables IV to VI provide some pertinent details.

SUMMARY

The ductus arteriosus with or without symptoms should be closed by operation if it remains patent beyond the age of 5, in order to avoid the hazards of retarded physical development, cardiac decompensation, or bacterial endocarditis.

A patent ductus is in effect an arteriovenous fistula within the heart and has the same effects upon the circulation as a peripheral arteriovenous fistula including increased cardiac output, limited in the case of a ductus to one-half of the heart, increased blood volume, reduced blood pressure, particularly diastolic pressure, cardiac dilatation, and eventually decompensation.

Cardiac failure may occur at an early age if the ductus is large and easily distensible. As the child with a patent ductus grows to adult stature the lower resistance imposed by the pulmonary circulation as compared with that of the systemic circulation will attract a progressively greater volume of blood into the shorter circuit, resulting in progressive dilatation of this shorter circuit including the ductus and the left heart. This progressively increasing blood flow through the shorter circuit is limited by two factors, the innate rigidity of the ductal wall itself and the increasing fibrosis of the pulmonary capillaries incident to their connection with the high pressure of the systemic circulation. These two factors diminish the difference in the resistances of the two systems and reduce, and may even abolish, the tendency for an increasing volume of blood to flow through the ductus, the effects of which then remain stationary. Both factors are operative in delaying the appearance of cardiac symptoms to adulthood, but the appearance of cardiac symptoms is inevitable if a difference in the resistances of the two systems persists, if the ductus remains open, and if its walls remain distensible. Just as a dilating peripheral fistula eventually results in cardiac decompensation, so will a dilating ductus increase the flow through it and produce left heart failure.

Bacterial endocarditis and endarteritis occurred in one patient at the early age of 7, cured by ligation of the ductus. An aneurysm of the ductus occurred in another patient of 11 years whose ductus had been ligated twice previously and was accompanied by a bacteremia completely resistant to all antibiotics. Division of the ductus at the site of the aneurysm resulted in immediate disappearance of the bacteremia and complete restoration to health.

In another patient of 14 years of age, an aneurysm of the pulmonary artery opposite the entrance of the ductus followed a bacterial endarteritis for which 992 injections of penicillin had been given with control of the bacteremia. Ligation of the ductus and wrapping of the aneurysm with Cellophane has stayed the aneurysmal process.

A pair of twins, 3½ years old, showed about the same clinical response to patent ductus both before and after an operation in the course of which remarkably similar ductus in shape and size were found. The circulatory effects of one ductus were slightly more pronounced as shown by a larger heart and a lower diastolic pressure. At operation this ductus measured one millimeter larger in diameter at both the aortic and pulmonic ends.

One patient, cyanotic in life, was not operated upon due to a mistaken diagnosis, and died of cardiac failure at the age of 16 years. Necropsy revealed evidence that the flow of blood through the ductus throughout life had been from pulmonary artery to aorta with an excessive development of the right heart as compared with the left. It is questionable if the patient could have survived ligation of the ductus at this age because of the preponderant hypertrophy of the right heart. Recognition of this condition earlier in infancy would no doubt have permitted permanent correction by operation.

A woman 26 years of age with a known patent ductus but without serious symptoms for many years became pregnant. At the end of about a month, increasing dyspnea and easy fatigability led to division of the ductus, which eliminated all cardiac symptoms. This experience provides added support for early operative closure of a ductus no matter how symptomless it may be in childhood.

Five patients with all the signs and symptoms of patent ductus were operated upon by thoracotomy but no ductus was found. One patient, a married woman, 25 years of age, died following thoracotomy with advanced signs of bacterial endocarditis. At necropsy, a high interventricular septal defect with an aneurysm of the sinus of Valsalva communicating with the pulmonary artery, complicated by the extensive vegetations of a bacterial endocarditis, was found. The other four patients recovered without accentuation of symptoms and were presumed to have aortic-pulmonic septal defects or high interventricular septal defects involving both aorta and pulmonary artery.

Eight patients with patent ductus were treated by division and sixty-seven by multiple ligation in continuity. The first ductus operated upon recurred following ligation with one ligature, and one ductus recurred following triple ligation with the formation of an aneurysm. Both patients were cured, the first by religation, the second by division of the ductus at the site of the aneurysm. One death, in seventy-five cases, occurred on the third day following operation for division of the ductus due to a complete anuria incident to blood transfusion.

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DISCUSSION

DR. RICHARD H. MEADE, Grand Rapids.—I have no intention of discussing this excellent paper as I am not qualified to do so, but I do wish to report an interesting experience that my associate, Dr. Rasmussen, had two years ago, as it may be of value to some of you.

The patient was a woman in her 20's who had an obvious patent ductus arteriosus. At operation, during the dissection about the duct, serious bleeding was encountered which necessitated ligation of the pulmonary artery in order to control it. The ligation was done without fear because we knew that it had been demonstrated that it produced no deleterious effects. A few days later we left for the Denver meeting of this Association with no worries about the patient. Upon our return, however, it was evident that she had gangrene of the lung and a pneumonectomy was done. She died at the conclusion of the operation. Autopsy failed to demonstrate any abnormalities that could have accounted for the unusual course of events.

It is our belief that the gangrene developed because this patient's lung had been supplied for some twenty years with arterial blood through the ductus, as well as through the bronchial arteries. The bronchial arteries had, therefore, not developed normally and when the entire load of the circulation had been thrown on them by ligation of the pulmonary artery they had been unable to meet the demand. It is likely that an infant could tolerate ligation of the pulmonary artery, but we believe that should it ever become necessary to ligate the pulmonary artery in an adult with a patent ductus arteriosus, then the lung also should be removed.

DR. EDGAR W. DAVIS, Washington, D. C.—I should like to make a few comments on the presentation of Drs. Holman and Gerbode, which I have enjoyed immensely. Upon many occasions, at the meetings of this Association, when discussing the technical procedure of double ligation versus division and suture of patent ductus arteriosus, I have defended the former method. Several years ago, when I discussed papers by Drs. John Jones, Robert Gross, and Clarence Crafoord, I reported a series of twenty-five or more patients upon whom I had operated, using the multiple ligature method, and I stated at that time that we had had no recurrences and no serious complications. On that basis, I saw no reason to change to the technique of division and suture.

Now, after a period of additional years, with accumulated experience involving more than a hundred cases, I find that we have two definite cases of recurrence or recannulization, and possibly a third case which at this time has not been proved. The patent ductus clamp designed by Dr. Willis Potts has made the operation of division and suture one of complete safety. As a matter of fact, out of our first sixty-five patients operated upon, we did double ligations in sixty-three and divided and sutured in only two. Because of the fact that the operation can now be performed with safety with the use of the Potts clamp, thereby minimizing the percentage of recurrence, I wish to take this opportunity to retract my previous statements in which I advocated double ligation instead of division and suture. So, in the words of the famous poet, Edgar Allen Poe—

"Be not the first by whom the new is tried,
Nor the last to lay the old aside."

DR. HARRIS B. SHUMACKER, JR., Indianapolis.—The fascinating paper of Drs. Holman and Gerbode has raised a great many interesting problems. I propose to discuss only one of them, that of right ventricular hypertension in cases of patent ductus arteriosus. We have had three patients whose disorder fell into this category. In one child the right ventricular pressure was approximately the same as the systemic arterial pressure. Unfortunately, in this case studies were not complete enough to permit us to say whether there was reversal of flow through the ductus during any phase of the cardiac cycle. At the time of operation the ductus was dissected free and clamped cautiously. Marked tachycardia developed and the clamp was released. On several successive occasions clamping the ductus brought about the same alarming increase in cardiac rate. Finally, however,

the ductus could be clamped without change in rate or rhythm of the heart. The ductus was consequently divided. During the process of suturing the divided ductus the patient developed auricular fibrillation. Fortunately she withstood the operation without difficulty. Shortly afterward conversion to a normal rhythm was achieved, and she has remained quite well.

In one young woman in her late 20's there was good evidence of reversal of flow through the ductus from cardiac catheterization studies and from study of arterial oxygen saturation in upper and lower extremities. Because we were informed of several disastrous attempts of others to treat cases of this sort surgically, we elected not to operate upon this patient.

From cardiac catheterization, a third patient, also in her late 20's, was known to have a large atrial septal defect in addition to a patent ductus. At times the right ventricular and pulmonary artery pressures were higher than that of the aorta and there was good evidence of reversal of flow through the ductus during some phase of the cardiac cycle. There was additional evidence of some unexplained vascular shunt in the lungs. Because of this fact and the presence of an atrial septal defect, it was thought that she might withstand division of the ductus. The hazards of operation were explained to the patient and she desired to have us proceed with it. When the ductus was dissected free it was clamped with caution. There was no change noted in cardiac rate or rhythm, arterial or venous pressure, and there was no evidence of pulmonary edema. Accordingly, the ductus was divided. Her convalescence was uneventful. Postoperative catheterization studies reveal the right ventricular and pulmonary artery pressures still to be very high. In contrast to the situation before operation there is now evidence of a left to right ventricular shunt. It thus appears that this patient had a ventricular septal defect in addition to an atrial septal defect and a patent ductus arteriosus. She apparently has a complicated and belatedly recognized Eisenmenger complex. If there has been any change since operation it has been one of mild improvement.

DR. PAUL T. DE CAMP, New Orleans.—I should like to refer to two patients with persistent patent ducti, pulmonary hypertension, and a partial reversal of blood flow through the ducti. To date, there is no adequate explanation for the increased intrapulmonary resistance and the pulmonary arterial hypertension which develops in some patients with patent ducti. There are a few reported cases, similar to the one reported by Dr. Holman, in which there finally develops a complete reversal of flow with generalized cyanosis. In the presence of significant pulmonary hypertension the clinical diagnosis of patent ductus arteriosus is difficult, because the diastolic phase of the typical continuous murmur tends to disappear. The diagnosis may be clarified by retrograde aortography and cardiac catheterization. In our opinion, as long as the degree of shunt from the aorta to the pulmonary artery exceeds the shunt from the pulmonary artery to the aorta, the patient may be considered as a candidate for ductal obliteration. The necessary information may be computed from the determination of the cardiac output and the oxygen content of simultaneous blood samples obtained from the right brachial, a femoral, and the pulmonary artery.

One patient, an 11-year-old girl, had had serious cardiac symptoms for several years. Congestive failure was controlled with difficulty. The clinical diagnosis was difficult, and the diagnosis was established only by retrograde aortography and cardiac catheterization. The pulmonary arterial pressure equalled the aortic pressure. The left to right shunt slightly exceeded that from right to left. At operation, fifteen minutes after the application of clamps to the ductus, a peculiar type of cardiac arrest occurred. Regular auricular and ventricular contractile movements continued but no blood was expelled from the heart because diastolic relaxation did not occur. Cardiac massage was ineffective. Normal cardiac activity resumed after six minutes and the ductus was quickly ligated, but the child died six hours later from the results of cerebral damage.

A 32-year-old man has had known heart disease all his life with reduced activity and retarded physical development. Hemoptysis was present for ten years and culminated after

hospital admission in massive hemoptysis of 1800 c.c. of blood in a twenty-minute period. Pulmonary hypertension, a patent ductus, and pulmonary arterial sclerosis were diagnosed on clinical, electrocardiographic, and roentgenologic examinations. Following transfusion therapy and the demonstration, on bronchoscopy, of a blood clot protruding from the right middle lobe orifice, an emergency right middle lobectomy was performed. A pulmonary arterial aneurysm 1.5 cm. in size was found, which had ruptured and filled the entire lobe with blood. The patient has recovered from the lobectomy. Studies of the degree of the shunt through the ductus have not been completed, but we have evidence of a partial reversal of blood flow.

DR. HOLMAN (Closing).—I am very grateful for these very fascinating additional aspects of this problem, particularly Dr. Shumacker's remarks. I would like to say that when we ligate the ductus we do not rely on two ligatures. We try to emulate Dr. Blalock by applying two ligatures at either end of the ductus and two transfixing ligatures between them, with frequently a fifth at the center of the ductus. I am sure that two ligatures will not suffice. We have divided the ductus in eight cases. We recently divided the ductus in a child aged 6, who had a ductus 1.5 cm. in diameter. Ligatures around such a large ductus can easily cause trouble with fracture of its walls. But we are content in most instances to do the quadruple or quintuple ligation.

The problem that Dr. Shumacker raises is, of course, an interesting one, and it shows the value of first attempting to find out by digital pressure on the ductus, whether the patient will tolerate ligation of the ductus, or not. Occasionally, a ductus may be encountered which provides flow into the lung in a mild case of tetralogy of Fallot, and under these circumstances one would not want to ligate it. This question of tachycardia that yielded eventually to closure of the ductus, probably has to do with reversal of flow through the ductus. Of course when we have increased pressure in the pulmonary system incident to a direct connection with the aorta, pulmonary fibrosis and pulmonary hypertension is the inevitable result, and it may be that peripheral resistance in the two systems will eventually equal each other, with the result that flow through the ductus will alternate, first in one direction and then in the other, depending upon such other factors as complete rest or violent exertion.

THE ANATOMIC (HISTOLOGIC) BASIS AND EFFICIENT CLINICAL SURGICAL TECHNIQUE FOR THE RESTORATION OF THE CORONARY CIRCULATION

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THE principle of arterialization and of the reversal of the flow of blood through the coronary sinus and its ramifications was first suggested by Roberts[1] and was first successfully carried out in both the experimental laboratory and in human clinical cases by Beck and his co-workers.[2-5] It is the purpose of this article to confirm the beliefs of Beck and associates and to establish the anatomic basis for the clinical benefits observed. A relatively minor technical modification of the original Beck operation is offered which is believed to lessen the likelihood of thrombosis and obliteration of the arteriovenous anastomosis.

The chief field of applicability of this procedure is found in the treatment of occlusive disease of the coronary arterial system (atherosclerosis). However, arterialization of the coronary sinus might well prove useful in individuals presenting the anomaly of one or both coronary arteries (especially the left) arising from the pulmonary artery.

Coronary occlusion, myocardial infarction, angina pectoris, and acute coronary insufficiency are all essentially manifestations of the same underlying process—arteriosclerosis affecting the coronary arterial system. Like arteriosclerosis elsewhere, it may be part of a generalized process involving all of the arteries of the body in approximately equal degree, or it may be so much more advanced and progressive in the coronary arteries than elsewhere as to seem to be an isolated process. It is essentially these latter cases which offer the best indications for surgery and the best clinical results.

From the clinical standpoint, one may consider coronary arteriosclerosis to be essentially an occlusive process in which the lumina of the large and of the medium-sized coronary arteries are progressively diminished by subintimal thickening of the vessel walls. Plaques of subintimal cholesterol deposition or of actual ulceration may be seen predisposing to the development of thrombosis with complete obliteration of the lumen (Fig. 1). Since the muscle-bearing moderate-sized branches of the coronary arteries are functionally similar to end arteries for a given area of the heart, the segments of myocardium normally supplied by each of these vessels may become infarcted unless sufficient collateral channels have become established or have opened up to maintain nourishment of the myocardium. Schlesinger and Zoll[6] have

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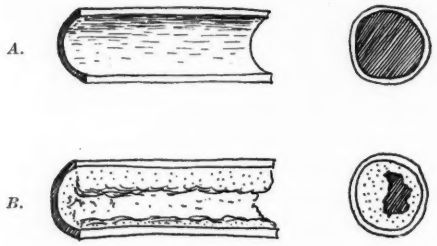
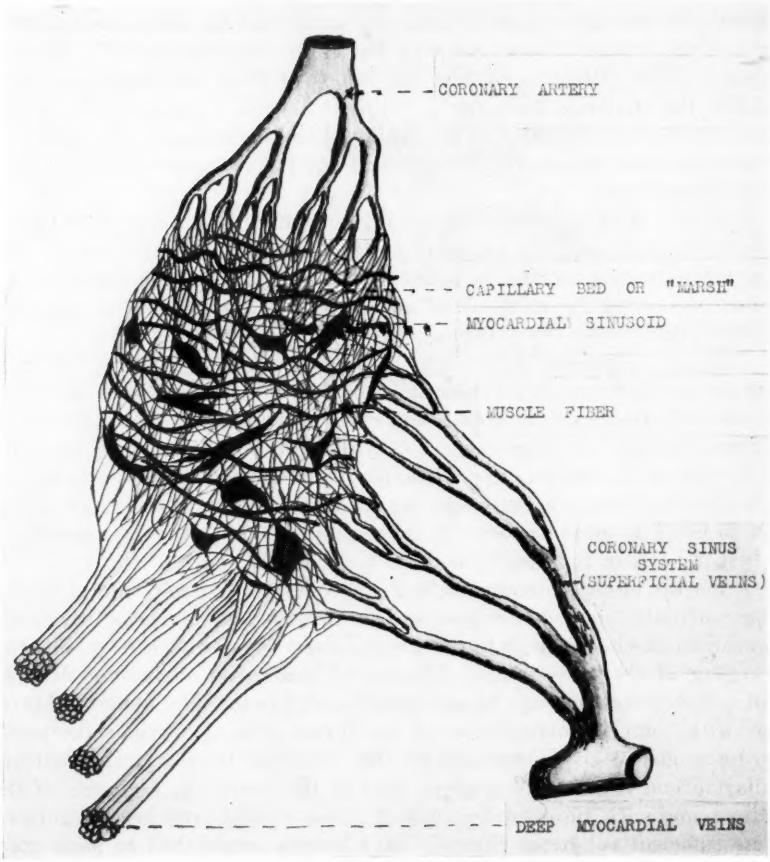
This work was supported by the Mary Bailey Foundation for Heart and Great Vessel Research. The anatomic work reported in this paper was supported by U.S.P.H. Grant H-321.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery at Dallas, Texas, May 8-10, 1952.





C.

Fig. 1.—*A*, Diagrammatic drawing of a normal coronary artery. *B*, Diagrammatic representation of the diminished lumen in a coronary artery produced by subintimal thickening of the vessel walls. *C*, Concept of left ventricular myocardium as a "marsh" fed by branches of the coronary arteries. After depletion of its oxygen, 40 per cent of the blood is drained off by the deep myocardial veins and 60 per cent is drained by the "expendable" coronary sinus system. The latter therefore offers the surgeon an available, even more extensive system of blood vessels capable of transporting arterial blood to the capillary bed in cases with diminution in coronary arterial flow.

shown that only the larger and medium-sized branches of the coronary arteries are affected by this process, the smaller branches without muscular coats being spared. Hence there are some possibilities for the development of an adequate collateral blood supply through these smaller branches. However, the development of such collateral circulation (intercoronary communications) is often a slow process and is inadequate to protect against the effects of a sudden complete occlusion of one of the coronary arteries.

At best, the development of such collateral circulation between the various segments of the coronary arterial system is unpredictable, due to the marked variation in the vascular pattern in different individuals. It can improve the distribution of the blood provided by the major coronary arteries, but it cannot significantly increase the total amount of blood available for myocardial metabolism and nutrition if there is appreciable diminution in this central coronary supply. Hence all operative and medical mechanisms which rely for their effects upon the development or enhancement of normally minimal intercoronary blood flow are limited in their ultimate clinical potentialities. Only those processes and procedures which have the effect of augmenting the total coronary circulation can be effective when there is a progressive and severe diminution in the total myocardial flow.

Such possible augmenting processes include the overdevelopment of normal intracardiac openings which might permit direct passage of blood from the chambers into the deeper myocardial sinuses (sinusoids), and the development of additional vascularity at the base of the heart, and in adhesions occurring between the heart surface and the adjacent pericardium (extracardiac circulation). Such natural compensatory processes may, in certain unusual individuals, entirely replace the normal coronary arterial flow when the latter gradually becomes completely obliterated.[7] However, these natural processes are relatively inadequate to protect completely the average affected individual from the effects of a sudden major coronary occlusion, and so cannot be relied upon.

Attempts have been made by Beck,[8] O'Shaughnessy,[9] and others to augment the effect of these natural mechanisms by suturing adjacent vascular tissues (omentum, lung, pericardium, pectoral muscle) to the surface of the heart. Beck has made a point of removing the epicardium with a burr in order to eliminate this possible anatomic barrier to adequate vascular communion between the graft and myocardium.

Thompson and Raisbeck[10] have used powdered talc in order to produce vascularized adhesions between the pericardium and the epicardium. Beck and co-workers,[11] after extensive animal experimentation, suggested the use of powdered asbestos which they considered more effective than talc. Asbestos not only stimulates the production of vascularized adhesions between the epicardium and the pericardium, but leads to an extensive dilatation and overdevelopment of normal intercoronary communications. It is interesting that some patients so treated seem to have been improved.

It is our considered opinion that only a very small percentage of the normal coronary flow can be replaced by such extracardiac sources. Thus

these measures really afford only partial protection against immediate death from a sudden arterial occlusion, and a certain measure of possible prolongation of life and comfort. For these reasons, Beck[2] has inevitably turned his attention to the possibilities of largely or completely replacing the normal coronary blood flow. He hopes thus to permit a complete or appreciable restoration of the patient to health and activity. He has experimentally supplemented the coronary arterial flow by shunting arterial blood through the coronary sinus into its tributaries.

There are those who question the possibility of retrograde blood flow through the coronary sinus system into the myocardial capillary bed. We propose to show that this flow can be reversed when arterial blood is shunted into the coronary sinus, at least after subsequent narrowing of the sinus near its exit into the right atrium has been accomplished.

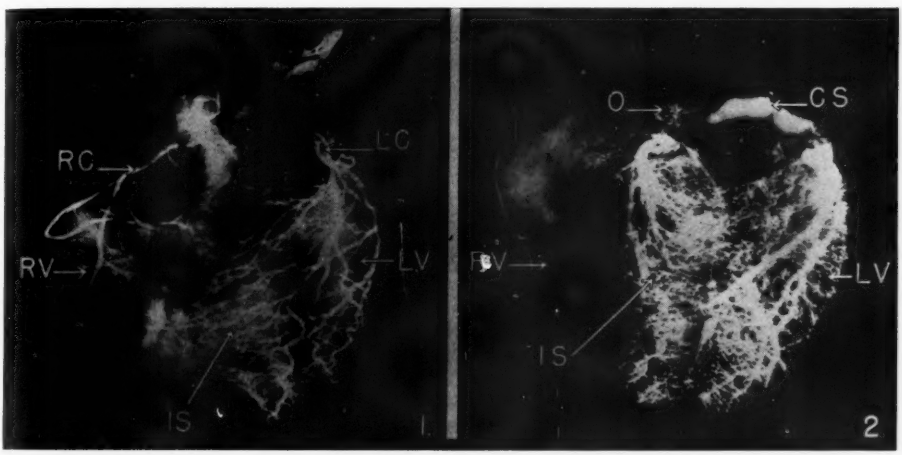
Others[12] have intimated that natural communications exist between the superficial cardiac and the deeper myocardial veins which drain directly through small openings into certain of the cardiac chambers. Such communications would permit arterial blood entering the coronary sinus to escape by way of the myocardial veins. Such blood would be lost from the capillary bed and hence could not nourish the cardiac muscle. We are aware that such communications exist[13] and feel that they afford a valuable safety mechanism regulating the retrograde flow. They do not defeat the physiologic purpose of the operation. While some of the blood admittedly escapes through them into the right atrium, a residual quantity may be distributed through the tributaries of the coronary sinus into the capillary bed of the myocardium. The amount of this capillary flow may be proportional to the deficiency in supply from the coronary arteries. Should too great a quantity of blood enter the sinus by virtue of the establishment of a very large graft, these communicating veins will undoubtedly permit the excess to drain off into the cardiac chambers. Thus, engorgement and damage to the myocardial capillary bed are avoided. Furthermore, these communications, especially those involving the epicardial veins, represent the only possible route for getting blood from the coronary sinus to the right ventricular myocardium.

ANATOMY

Recent anatomic studies by one of us (R. C. T.) have revealed some information on the coronary circulation which is of considerable significance in the Beck procedure.[14] By means of wax model reconstructions of the human newborn heart, it was possible to demonstrate continuity between the large veins and venous sinuses of the ventricular myocardium and the lumen of the coronary sinus. Similar continuity was demonstrated between the myocardial venous channels, the lumen of the right atrium, and the cardiac veins lying in the epicardium. It is interesting to note that many of these thin-walled venous sinuses were found to accompany and to ramify in the common bundle of conduction tissue as they crossed the fibrous ring of the heart.

These microscopic observations on the venous supply of the heart have more recently been verified and extended by the usage of injection experi-

ments, radiographic films, and study of serial celloidin sections (Angulo and Truex[15]). These authors studied both the arterial and venous systems of the human and dog heart with a standardized barium sulfate-gelatin solution. By comparing the arterial and venous injected specimens they clearly demonstrated that, in the adult, the left ventricle and interventricular septum possesses predominant arterial and venous systems, whereas the right ventricle has a more meager vascular supply (Fig. 2, *A* and *B*). Greater significance is attached to their discovery that, within the left heart, the venous tributaries of the coronary sinus form an extensive network which is several times more abundant than that formed by corresponding branches of the left coronary artery.[13] This rich venous plexus, formed by tributaries of the coronary sinus, has been found to be continuous distally with the myocardial capillary bed.[16]



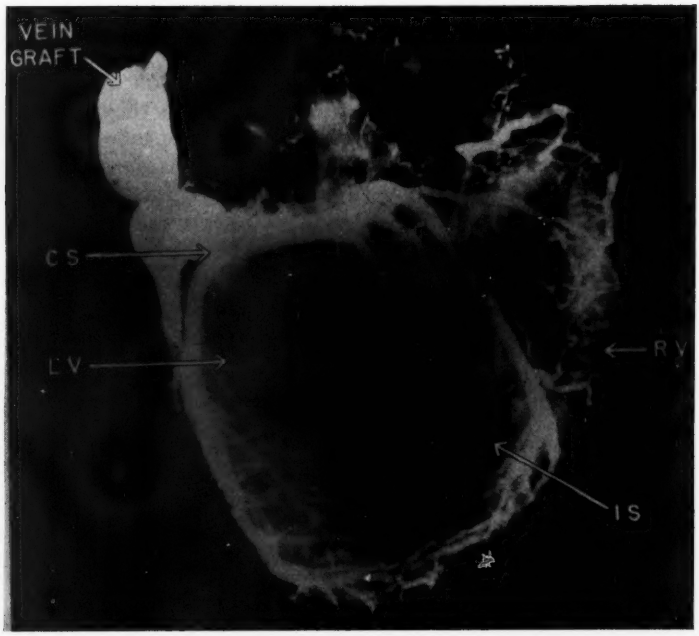
A. *B.*

Fig. 2.—*A*, Injection study of a human heart through the coronary arteries. *B*, Injection study of a human heart through the coronary sinus system. This reveals the numerous venous channels throughout the left ventricle and the interventricular septum. Note the small amount of filling over the right ventricle. (Courtesy of Wistar Institute.)

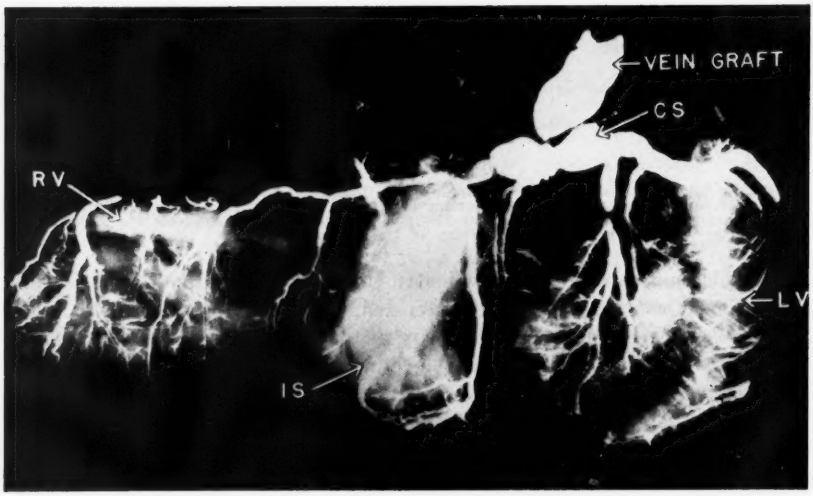
Hence, this anatomic work has indicated that when the normal arterial system of the heart becomes partially obliterated, as in atherosclerosis, the venous tributaries of the coronary sinus can by surgical intervention provide an alternate route to the capillary bed to meet the functional demands of the left ventricle and septum.

Injection studies have been completed on three dog hearts following the Beck procedure. All of these specimens demonstrate that following their arterialization, the branches of the coronary sinus establish large epicardial anastomoses with the small cardiac and anterior cardiac veins of the right ventricle. Through these anastomoses the right ventricle may also receive arterial blood after the Beck procedure (Fig. 3, *A* and *B*).

The vascular connections of the coronary sinus have also been studied.[13] It possesses anastomoses with the cardiac veins of the right ventricle; myo-



A.



B.

Fig. 3.—*A*, Injection study of a dog's heart following a Beck procedure. The injection media has been introduced by way of the vein graft into the coronary sinus. *B*, The same heart, opened after the method of Schlesinger. Note the vascular channels demonstrated throughout the heart and especially those of the right ventricle, due to the opening up of epicardial anastomoses by the pressure-head of blood entering the coronary sinus by way of the aorta. Compare with Fig. 2, *B*.

cardial veins and venous sinuses of the atrium, interventricular septum, and the left ventricle; and with the lumen of the right atrium. These collateral channels of the coronary sinus serve as a safety valve mechanism after the Beck procedure. Such collateral channels also return the major outflow to the lumen of the right atrium as indicated by their word schema (Fig. 4).

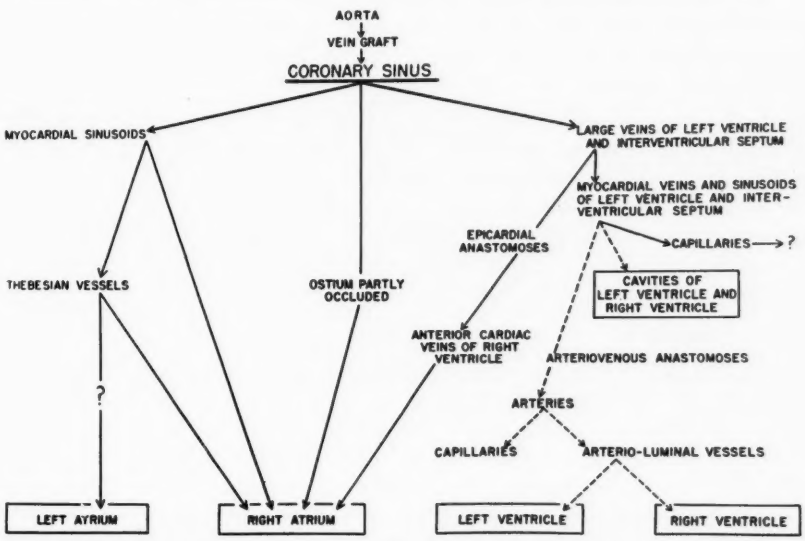


Fig. 4—Schematic representation of the pathway of blood entering the heart following a Beck procedure. The solid lines represent pathways which we have demonstrated. The broken lines represent pathways which are either theoretical or have been reported by others but which have not been demonstrated by us.

RESULTS OF ANATOMIC INVESTIGATIONS

Radiologic films and microscopic methods all demonstrate that:

1. The left heart and septum possess a much richer arterial and venous system than the right heart.

2. In the left ventricle and septum the venous system is far more abundant and extensive than the arterial system. This extensive venous network is drained by tributaries of the coronary sinus.

3. The coronary sinus and its tributaries possess continuity with the capillary bed of the left ventricle and septum. The coronary sinus also has several collateral channels leading to the lumen of the right atrium.

4. Arterialization of the coronary sinus by the Beck procedure permits arterial blood to enter the capillary bed of the left heart by retrograde flow through tributaries of the coronary sinus.

5. Following its arterialization, branches of the coronary sinus establish epicardial anastomoses with the small cardiac and anterior cardiac veins of the right ventricle. Through these anastomoses, the right ventricle also receives arterial blood after the Beck procedure.

6. Collateral venous connections of the coronary sinus serve as a safety valve mechanism after the Beck procedure. These collateral channels also return the major outflow to the lumen of the right atrium.

ANATOMIC CONCLUSIONS

Anatomic findings, to date, indicate that the Beck procedure offers vascular benefits to the entire heart in cases of advanced coronary arteriosclerosis. Better nourishment of the myocardium is attained by a combination of the following two factors:

1. Retrograde flow of arterial blood through the extensive venous tributaries of the coronary sinus provides new avenues to the capillary bed as well as drainage routes back to the heart chambers.

2. Development of anastomoses between branches of the coronary vessels and extracardiac arteries occurs at the base of the heart with the arteries of the pericardium, vasa vasorum of the aorta and pulmonary vessels, and new arterioles which grow into the wall of the vein graft.

PHYSIOLOGY

Physiologic studies in animals or patients upon whom arterialization of the coronary sinus has been performed are extremely few. Eckstein,[17] so far, seems to be the chief investigator to consider this problem.

In a series of dogs subjected to division of the left circumflex coronary artery before, and at varying periods of time subsequent to, performance of the first and second stages of the Beck operation, Eckstein was able to show dramatic changes in the amount and type of coronary flow from the cut end of the distal segment. The average changes are illustrated in Chart 1.*

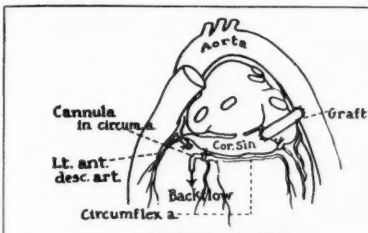
To simplify these findings, we may say that in the normal dog the blood flow from the cut end of the distal segment of the divided left circumflex coronary artery will average 2.5 c.c. of arterial blood per minute. This is presumably derived from the normally existing minute intercoronary anastomoses which bring blood from the remaining intact coronary trunks to the severed one. When a vein graft is anastomosed between the coronary sinus and the aorta, and the sinus outlet is largely obstructed, this flow from the cut coronary artery rises to 20 or 30 c.c. per minute. But the blood is dark and essentially venous, indicating that it has passed through a capillary bed. Presumably it is derived from the arterial blood which has traversed the coronary sinus, its ramifications, and the capillaries by retrograde flow.

With the passage of time this blood from the distal cut coronary end becomes more and more arterial in nature until after twelve weeks it is fully saturated with oxygen. It is thought that this phenomenon is due to progressive enlargement in the intercoronary communications until they are able to replace fully the interrupted normal coronary arterial flow. Undoubtedly the development of such efficient collaterals would be slower and less reliable in human patients with presumably generally diminished flow in all major coronary arteries.

*This chart is used through the courtesy of R. Eckstein and D. Leighninger of Cleveland, Ohio. (To be published.)

Having established what we believe to be a sound, theoretical, anatomic, and physiologic basis for arterialization of the coronary sinus, it now remains to show its surgical or technical feasibility and its clinical effectiveness.

We are indebted to Dr. Claude Beck who, having developed this surgical procedure almost singlehandedly, freely offered us not only the fruits of his fifteen years of animal experimentation, but also the advantages of his personal participation and surgical skills. We were thus able to start a clinical program of surgery for coronary artery disease at Hahnemann Hospital in Philadelphia, Pa., and at St. Michael's Hospital* at Newark, N. J. Rigid



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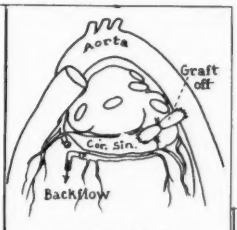

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TIME	BACKFLOW cc/min.	BACKFLOW cc/min.	DIFFERENCE IN OXYGEN CONTENT Vol.%
Normal dogs without arterialization (at time of placing graft sinus press. at 50 mm. Hg.)	7.6	2.8	10.9
At time of sinus ligation (3 wks. after placing graft)	20.8	5.6	8.2
1 week after sinus ligation	22.0	10.0	9.4
2 weeks	23.0	8.0	9.9
3 weeks	24.0	19.0	3.6
1 month	30.0	28.0	2.1
3 months	16.0	20.0	0.0
4 months	11.0	15.0	0.0
6 months	20.0	22.0	0.0

Chart 1.

requirements for the selection of patients for surgery were set up. As a result of this selectivity we were able to show an operative mortality up to the present time of only two cases of the eighteen patients operated upon. This record clearly proves that patients with serious impairment of coronary flow can withstand these operative procedures if the disease process is not too advanced. The operation is not an intracardiac one but merely a meticulous anastomosis performed between a systemic artery, the aorta, and the coronary sinus. This does not cause any severe physiologic disturbance of cardiac function even during the actual performance of the surgery. Therefore, there is no reason why the inherent operative mortality should be high.

*The patients operated upon at St. Michael's Hospital are included with the permission of Dr. Anthony Crecca, Chief of Thoracic Surgery.

SELECTION OF PATIENTS

Criteria for the selection of patients have been established. However, the present arbitrary standards undoubtedly will be modified by further clinical experience.

Disability resulting from atherosclerotic disease of the coronary arteries varies. It is imperative that the operation be performed only for a degree of incapacity that warrants major surgical interference. At the same time the operation should not be considered where the disease is so advanced that benefit or survival cannot be reasonably expected.

For this reason certain indications and contraindications as to operability have been used for the selection of patients. In the short medical history of coronary artery disease many facts have emerged as important prognostic guides. These have been utilized as the basis for further classifying the indications and contraindications as either major or minor.

Experience with the selection of cases for mitral commissurotomy has proved that clinical facts must be evaluated in the light of all attending circumstances. Classification schemes for operability are too inelastic to be practical. Few patients are entirely acceptable or unacceptable for surgery. Proper selection depends upon the critical appraisal of the indications and contraindications and an analysis of their respective importance in a particular patient.

Indications.—The basic requirement for surgery is major or progressive atherosclerotic disease of the coronary arteries. The diagnosis must be established beyond question and confirmed by definite abnormality in the electrocardiogram.

The functional impairment of the patient is the most important consideration. Anginal distress must be frequent and severe enough to interfere seriously with ordinary activity before it can be classified as major. Angina decubitus may be a particularly important indication. In all instances a reasonable trial with the usual medical routines should precede surgical treatment.

The presence of a previous myocardial infarction lends significance to the effort syndrome and increases the advisability of surgery. Where a myocardial infarction has not taken place, the most reliable history of anginal pain must be supported by proper abnormality in the electrocardiogram before it can be considered a major indication. Effort pain, however significant, without electrocardiographic abnormality does not establish the diagnosis of coronary artery disease beyond question, at least in relation to the surgical treatment.

The uncertainty of coronary artery disease, with its implied threat of sudden death, cannot be considered alone as the primary reason for operation at the present time. The basic requirement of disabling angina must be supported by evidence of a previous myocardial infarction or definite electrocardiographic abnormality before it becomes a major indication for surgery.

At this time it appears that patients who have electrocardiographic evidence of myocardial damage due to coronary artery disease, but who are not incapacitated, should not be considered as candidates for surgery even though they have had a previous myocardial infarction.

The significance of symptomatic coronary artery insufficiency is altered considerably by the age of the patient. The presence of the disease before the age of 40 is a major indication for operation. This probably holds true through the fourth decade. From that point, age ceases to become a major indication. Indeed, after 55 it acts as a deterrent.

Normal blood pressure and normal heart size increase the importance of any major considerations for surgery.

The various abnormalities which may appear in the electrocardiogram cannot be rigidly classified according to their importance. Those changes which appear constantly in the tracing probably represent more extensive pathology than those appearing after exercise. However, they may not represent a more significant prognostic guide.

Contraindications.—A recent myocardial infarction is a major contraindication. The operation should not be performed until six months have elapsed. Care must be taken that a severe, easily initiated effort syndrome does not indicate a developing infarction. When serial electrocardiograms show rapidly progressive myocardial damage, the operation must be deferred until the pattern has become fixed for four to six months. This applies even though there may have been no change in the patient's symptoms.

Two myocardial infarctions within one year is a major contraindication. This probably holds true for all patients with multiple infarctions regardless of the time interval.

Blood pressure values which remain above 180 mm. Hg systolic and 100 mm. Hg diastolic are considered firm contraindications. Evanescent readings beyond these levels are of lesser significance at this time.

Cardiac enlargement up to 10 per cent of predicted values is a minor deterrent. As the size increases thereafter this factor becomes of major importance.

The presence of congestive heart failure is an absolute contraindication. This is equally true when gallop rhythm is present. Heart failure that has responded to medical treatment still remains a major contraindication. It would appear that the degree of myocardial damage usually found in patients who have suffered congestive failure on the basis of coronary artery disease is too extensive to be aided by operative interference. However, undue cardiac enlargement, prohibitive hypertension, or multiple infarctions are more likely to be found in this group.

Certain associated diseases may serve as minor contraindications. These would include emphysema, bronchiectasis, diabetes, and marked obesity. Renal failure obviously would gravely compromise the surgical risk.

For technical reasons a markedly calcified aorta must be accepted as a distinct contraindication.

At the present time any single major contraindication is sufficient reason to deny surgery. Such issues as recent or multiple infarctions, congestive failure, gallop rhythm, gross cardiac enlargement, or even moderate hypertension cannot be counterbalanced by any major indication for operation. These patients are the least likely to survive or, if they do, the least likely to benefit.

The ideal candidate offers no major or even minor contraindications. The acceptable candidate may offer one or two minor deterrents which are of limited significance when compared with the major indications that are present.

Preoperative Management.—Patients who have been selected with care for surgery rarely present difficult problems in preoperative management.

A careful history will reveal the circumstances under which anginal seizures occur. Every attempt should be made to avoid duplicating these situations during hospitalization. Where necessary activity is restricted to a minimum, emotional tension is likewise reduced as much as possible. In this regard the period of laboratory and clinical investigation should be regulated according to the patient's tolerance and not to an inviolate routine. Brief words of explanation often sustain a patient through the most trying type of study.

Nitroglycerin is an invaluable drug, and there is no contraindication to its use in the preoperative period. Occasionally the so-called coronary vasodilators may aid in the control of frequent cardiac pain. Rest and mild sedation, however, appear to be the most effective measures before surgery.

Major arrhythmias are unusual in this group of patients. Premature ventricular systoles and the implied threat of ventricular fibrillation are best controlled by oral quinidine or Pronestyl Hydrochloride. These drugs are discontinued at least twenty-four hours before surgery since they may potentiate the effect of the intravenous procaine which is used routinely during the operative procedure.

There is no purpose in digitalization prior to surgery. Indeed, the drug may be harmful in initiating troublesome arrhythmias or conduction defects. Where this drug has been given in the past it should be discontinued unless a pressing indication for its use is still present.

Postoperative Management.—The major cardiac sequellae which develop after the first stage procedure are a persistent sinus tachycardia, premature atrial contractions, or atrial fibrillation. The sinus tachycardia requires no specific therapy. It does not respond to digitalis and usually subsides on the fourth or fifth postoperative day. Frequent premature atrial contractions usually precede atrial fibrillation. The latter, however, has occurred with no previous indication of atrial irritability. When the ventricular response to atrial fibrillation is uncommonly rapid, and the integrity of the circulation threatened, or when congestive failure actually develops, digitalis or any of the related compounds may be given. If failure is not present, however, the drug should be withheld if possible. The rhythm will revert spontaneously during the first week. Occasionally quinidine has been given under these circumstances but the effect is questionable. One patient (A. D.) continued to show evidence of atrial irritability for two weeks after both the first and second stages even though full doses of quinidine were given.

Oxygen is a routine medication for the first twenty-four hours. It is rarely required for a longer period of time. The patient is encouraged to cough from the start. Retained secretions are promptly treated with aspira-

tion, bronchoscopy, or tracheotomy as indicated. The patients are fed a soft diet within twenty-four hours and are encouraged to feed themselves as soon as is feasible thereafter. This applies to either stage of the operation.

Attention to bowel function is usually deferred until the third day when a warm saline enema may be given if necessary.

Ambulation depends upon the febrile reaction, cardiac rate, and the general condition of the patient. Generally it is not attempted until the seventh to tenth postoperative day. Frequent leg movements in bed can alleviate the discomfort and inherent danger of too early activity in the cardiac patient. When walking is finally permitted, it is regulated to the patient's tolerance and not to a set routine.

Antibiotics are routinely used. Penicillin and terramycin have been favored. There is, however, no need for anticoagulant therapy. Patency of the graft apparently depends upon technical factors and not upon the incoagulability of the blood. The routine use of aminophyllin by vein may have a beneficial effect upon the coronary circulation without greatly increasing cardiac output or the danger of arrythmia.

Immediately after the first stage operation a to-and-fro bruit may be heard at the second left interspace anteriorly or high in the interscapular area to the left of the spine. Even though the graft remains patent, this murmur may become considerably diminished on the third or fourth day. It then generally reappears and remains constant. Tilting the patient to the right at times increases the intensity of the sound. It is generally best heard after a full expiration. No other murmur develops as the result of the surgery.

The postoperative period is usually well tolerated. Patients have shown no reluctance to enter the second stage after successfully withstanding the discomforts of the first.

TECHNIQUE

First Stage.—The original Beck technique as applied to human beings is as follows: A segment of a large superficial vein, preferably without valves, is removed under local anesthesia. Either the median basilic vein of the arm or the external jugular vein is usually chosen. If a valve-bearing segment cannot be avoided, the vessel must be marked so that the proposed flow of blood will be in the direction of the valve. All branches to the vein must be carefully ligated, and any excess adventitia or perivascular tissue is carefully dissected away. The segment is irrigated with heparin solution and placed between pads of moist gauze until needed.

Preanesthetic preparation,* medication, and anesthesiologic care are directed toward (1) decreasing the irritability of the heart, and (2) lessening any factors such as nausea, coughing, or straining that may be present just before or during operation or in the immediate postoperative period.

Preliminary medication is aimed toward having the patient rested, and the metabolic rate as nearly normal as possible without depression. Pentobarbital sodium (Nembutal), 0.10 to 0.2 Gm. (1½ to 3 gr.), or seco-barbital

*The anesthesia for these patients has been managed by Dr. Kenneth Keown of the Department of Anesthesiology at Hahnemann Hospital.

(Seconal Sodium) in the same dosage is prescribed the night before operation. From an hour to an hour and one-half before anesthesia, 0.1 Gm. of pentobarbital sodium (1½ gr.) is given by mouth, and meperidine hydrochloride (Demerol) 75 to 100 mg., with atropine sulfate, 0.60 mg. (1/100 gr.) are given intramuscularly. Shortly after administration of these drugs, a vein in the forearm or wrist of the right arm is cannulated under infiltration anesthesia.

After the patient is brought to the operating room, absolute quiet is essential. The equipment for making electrocardiographic tracings and determinations of blood pressure is applied after the patient has been placed in a supine position on the operating room table.

Ten minutes before induction of anesthesia, a solution of 0.2 per cent procaine in 5 per cent dextrose and distilled water is administered intravenously in the operating room. From 200 to 300 mg. of procaine is given before tracheal intubation is carried out.

Thiopental sodium (Pentothal Sodium) is then given intravenously in dosages sufficient to produce hypnosis (150 to 200 mg.), followed immediately by the intravenous injection of decamethonium bromide (Syncurine), 3 to 4 mg., and then 150 to 200 mg. more of thiopental sodium (Pentothal Sodium). By this time approximately 350 mg. or more of procaine has been given. A Magill endotracheal catheter with cuff is passed under direct vision by the oral route. A mixture of 50 per cent nitrous oxide and 50 per cent oxygen is administered from the time the patient loses consciousness.

During all steps apnea is avoided with its ensuing, although temporary, hypoxia. Respiratory function is augmented or controlled to insure adequate pulmonary ventilation with both adequate oxygen delivery and carbon dioxide elimination. The Beck-Rand respirator is most useful in these cases. Administration of nitrous oxide and oxygen in this concentration is continued throughout the course of anesthesia.

Procaine is continued at the same concentration of 0.2 per cent, with a basic flow of 1 Gm. per hour. Thiopental sodium is added as required for basal hypnosis. We believe analgesia is afforded by intravenous procaine.

At the completion of the anastomosis, the rate of flow of procaine is decreased. Usually thiopental sodium need not be added after the approximation of the chest wall has been started. We attempt to have these patients awake, moving, and responsive at the completion of surgery.

The usual curved posterolateral thoracotomy incision is placed over the left seventh interspace, being extended as far anteriorly as the midclavicular line. Bleeding points are ligated with fine silk. The seventh rib may be removed subperiosteally if added exposure is desired. Large fixed retractors are used to obtain exposure. The lung, if adherent, is detached from the chest wall, diaphragm, and mediastinum by blunt and sharp dissection, and the inferior pulmonary ligament is sectioned up to the inferior pulmonary vein. The lung is thereafter kept retracted upward by large warm gauze pads with or without the use of hand-held retractors. The diaphragm, if high or tending to move, is held down by gauze-covered, hand-held retractors.

The pericardium is opened from a point 3 cm. from the ventricular apex to the middle of the left atrioventricular groove, and then extended from this point to expose the left atrium and the termination of the inferior vena cava. The heart surface is inspected for evidences of previous infarction and the major coronary arteries are examined for sclerotic changes. All cut pericardial edges are supported by application of traction sutures at 2 cm. intervals (Fig. 5).

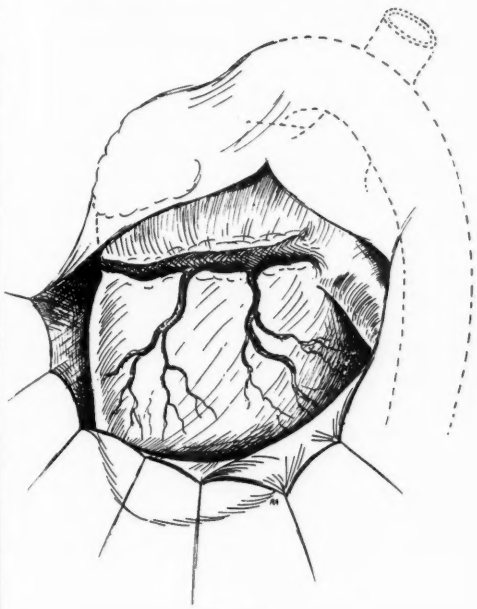

Fig. 5.

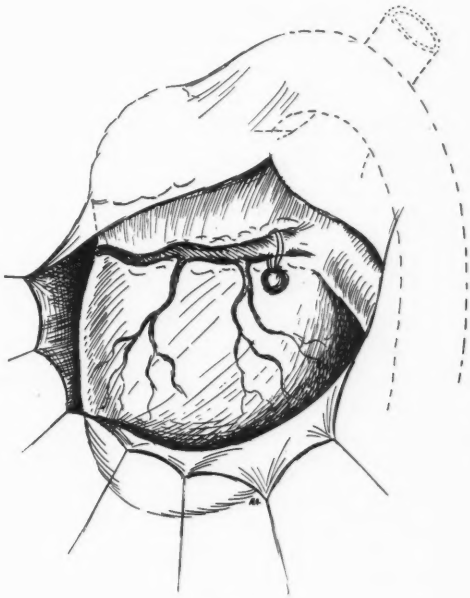

Fig. 6.

Fig. 5.—Exposure of the coronary sinus through the open pericardium.
Fig. 6.—The Orlon sutures are shown encircling the coronary sinus, ready for subsequent sinus ligation at the second stage.

The coronary sinus is identified and is traced to its terminus into the right atrium. It is partially dissected free from the epicardial fat in which it is somewhat imbedded, just above the point of termination. Traction sutures applied in the anterior lip of this epicardial fat may render the subsequent maneuvers easier. A curved half circle, one-inch needle is now threaded with a heavy plastic (Orlon) suture. The needle is passed completely around the sinus about 1 cm. from its terminus. Great care is taken that the needle does not puncture the sinus. The appearance of dark (unoxygenated) blood through the needle wounds is indicative of such puncturing, and requires that the suture be removed and the needle repassed. Actual passage of the needle through or around some of the muscle fibers of the atrial wall is acceptable.

This double suture is to be tied down at the second operative stage to obstruct partially (largely) the exitus of blood from the coronary sinus into

the right atrium. This step will ultimately insure the reversal of flow through the coronary sinus and its ramifications.

The needle is removed, and the ends of the sutures are matched up and tied together for subsequent ease of identification. They are then rolled upon a fine pair of hemostats, and a 00000 (arterial) black silk suture is passed through and around the tiny coil to maintain this compact and untangled state. The coil is then sutured out of the way under the heart and within the pericardium for easy accessibility and identification at the second operative stage, at which time reactive processes and adhesions will have largely obliterated identifying landmarks (Fig. 6).

A suitable site in the length of the coronary sinus is now selected for the anastomosis. This may be at any convenient point proximal to the previously applied Orlon suture. The apparent caliber of the sinus, its prominence and ease of mobilization, and its proximity to the thoracic aorta are all considered in the selection of the anastomotic site. Again the adjacent or imbedding fat is partially dissected away on either side of the sinus. Traction sutures may be used in the inferior (anterior) lip of fat.

Having partially mobilized the sinus in this region, two 00000 arterial silk sutures swedged upon atraumatic curved needles are passed longitudinally into the most prominent aspect of the sinus wall, taking bites about 3 mm. long, parallel to, and about 3 mm. apart from each other. Bleeding should be free from each needle puncture, indicating that the lumen of the sinus has been fully penetrated. If such bleeding is absent, the suture should be removed and replaced to prevent subsequent separation of the intima since this will cause difficulty in entering the sinus for performance of the anastomosis. Slight traction upon the stay sutures facilitates inclusion of a satisfactory amount of sinus wall for exteriorization within the jaws of the special curved sinus clamp (Fig. 7).

A No. 11 Bard-Parker bistoury-type scalpel blade (on an appropriate handle) is now used to make a longitudinal puncture opening in the excluded portion of the sinus midway between the two stay sutures (Fig. 8). This opening is extended in length for a distance of 6 or 8 mm. using a pair of angulated iris scissors. The size of the opening depends upon the size of the graft available for the anastomosis. The sinus lumen is then flushed with heparin solution.

A thirty-six-inch 00000 arterial silk suture with an atraumatic curved needle at each end is used to attach the end of the graft to the inferior (caudal) extremity of the incision in the sinus (Fig. 9). The suture is tied so that both ends are of equal length. One end is temporarily placed out of the operator's way while the other end is used to perform the anastomosis of the inferior (anterior) lip of the incision. This is accomplished by a continuous running, suture technique, the needle at each bite passing from the adventitial side of the graft, about 1½ mm. from the free edge, into the graft lumen, then from within the sinus through its wall and out the adventitial side. An attempt is made to place the sutures 1½ mm. apart in the graft. When the suture line reaches the longitudinal stay suture, the latter is removed. After

the suture line is completed, each stitch is individually picked up and tightened by the use of fine Potts type forceps. A separate stay suture may be placed at the upper (cephalic) extremity of the incision and tied to the end of the anastomotic suture. The suture line on the other side of the graft is similarly applied, and the free end is tightened and then tied to the end of the initial suture. Unless there has been puckering of the sinus opening, there is now a very adequate anastomotic communication between graft and sinus. The sinus clamp is removed permitting dark (venous) blood to run into the graft and out of its free extremity. The suture line is inspected

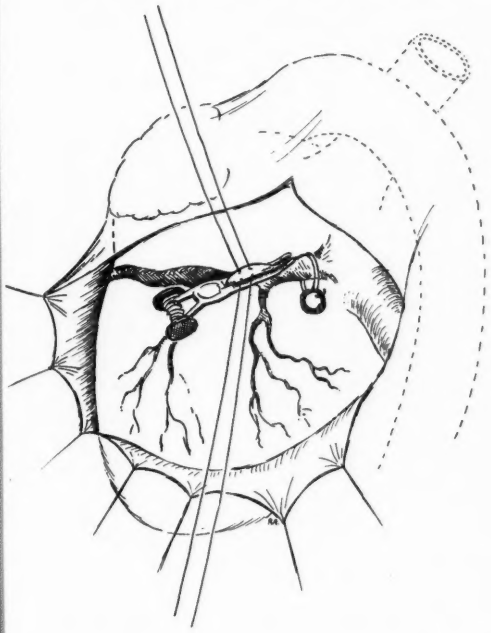

Fig. 7.

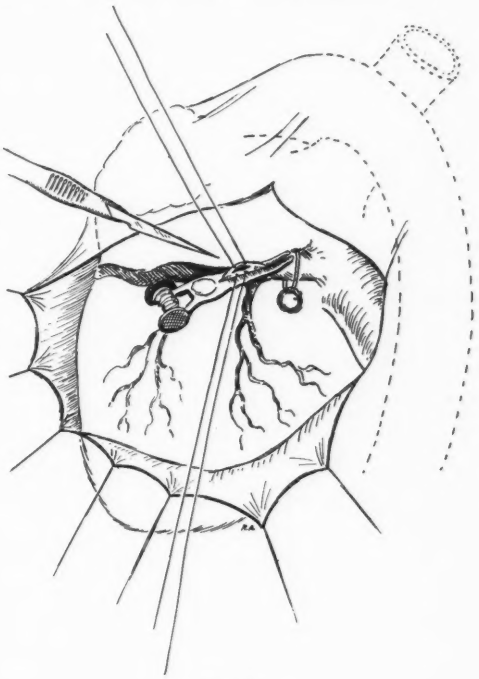

Fig. 8.

Fig. 7.—The site for anastomosis in the coronary sinus is tented by stay sutures, and a segment of the sinus is exteriorized by the clamp.

Fig. 8.—This shows the incision in the exteriorized segment of the coronary sinus.

for leaks and may be repaired by applying individual interrupted stitches if necessary. The graft is finally flushed with saline and heparin solution and is clamped off by an old-type rubber-covered bulldog clamp with a rather weak spring (Fig. 10).

Preparation is now made to anastomose the other end of the graft to the aorta. The graft is shortened to a length which is felt to be just adequate for a good anastomosis at approximately right angles to both the aorta and the sinus. All excess mediastinal fat and lymph nodes lying in the way of the graft are removed to avoid possible pressure upon and adhesions to the graft.

The selected site upon the aortic wall is mobilized. Two mosquito hemostats are applied to the aortic adventitia about 1 cm. apart, one cephalad and one caudad. As traction is made upon them, a special Beck toothed clamp is applied to the aortic wall. This clamp is designed to exclude a sizable portion of the aortic lumen while permitting blood flow beneath it. An attempt is made to exclude 1 cm. of the aortic circumference from the residual aortic lumen (Fig. 11). The mosquito hemostats are then removed and a portion of the adventitia is dissected and cut free from the site of the intended anastomosis.

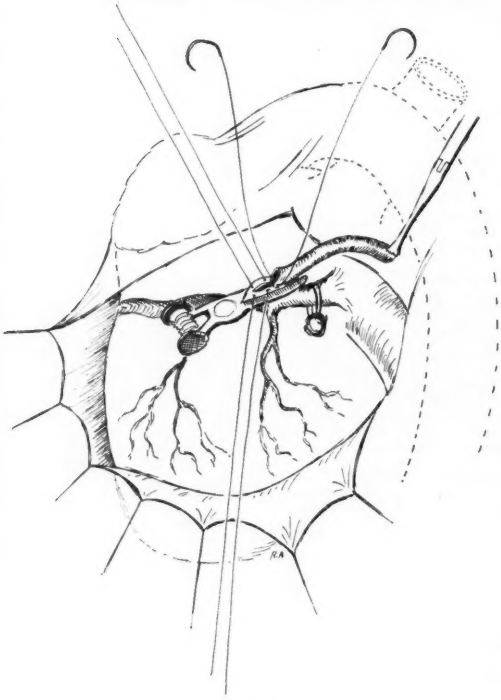

Fig. 9.

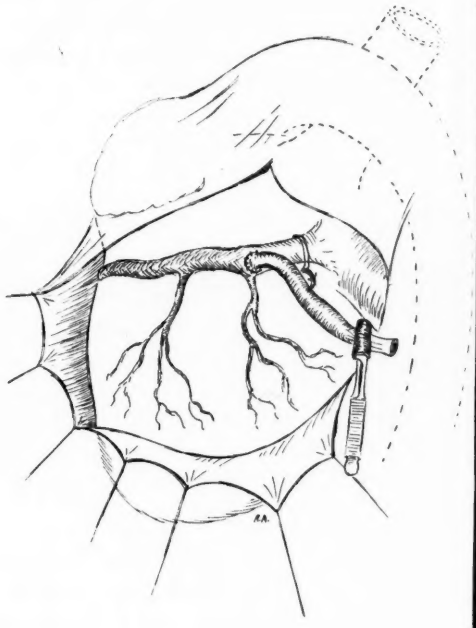

Fig. 10.

Fig. 9.—The beginning of anastomosis between the vein graft and the coronary sinus.
Fig. 10.—The vein graft is anastomosed to the coronary sinus, and the distal end is occluded by the small bulldog clamp.

Using a No. 11 Bard-Parker knife blade and handle, a 4 to 6 mm. longitudinal incision is made into the excluded portion of the aortic lumen (Fig. 12). Then using an identical technique, the free end of the vein graft is carefully anastomosed to the aortic incision. When it is completed, the bulldog clamp is removed from the graft and the suture line is examined for leaks. They may be controlled by individual arterial sutures, if necessary (Fig. 13).

The aortic clamp is gradually released and the suture line is again inspected. Arterial leaks may require only temporary gauze pressure for control or may require additional stitches. Sometimes the aortic clamp must be

replaced temporarily. After the flow through the graft is established, a vigorous continuous thrill is palpable throughout the graft and in the distal end of the coronary sinus. In persons with extremely impaired coronary arterial flow the red arterial blood from the graft may be seen immediately to extend a variable distance into the large veins draining into the coronary sinus. This effect can be produced or greatly enhanced by simply obstructing the outflow of the coronary sinus by traction upon the encircling Orlon sutures. With such traction all the cardiac veins are transformed in color from a blue to a distinct red.

Fig. 11.

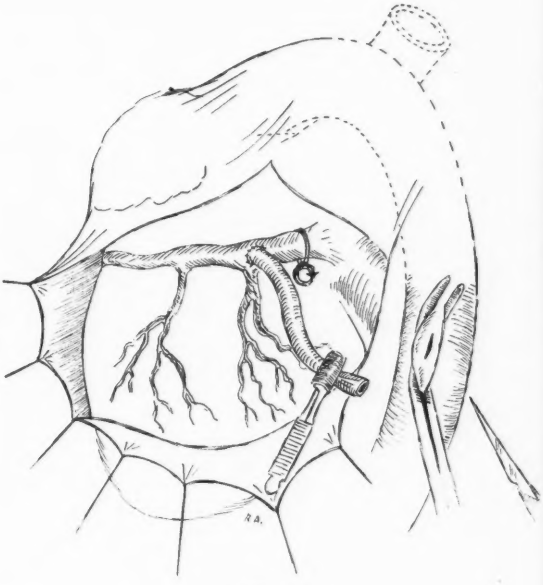

Fig. 12.

Fig. 11.—This shows a segment of the aorta tented by means of mosquito forceps and exteriorized by use of the special clamp.

Fig. 12.—Incision in the exteriorized segment of the aorta.

Second Stage.—Three weeks after the first operative stage, the patient is again anesthetized in much the same manner with an endotracheal airway. He is again positioned laterally, and the former incision is opened.

The lung is usually found to be adherent to the parietal wall, and a variable amount of loculated pleural fluid may be present. The lung is carefully mobilized in the lower thorax exposing the pericardium.

The previous pericardial incision is reopened, and any adherent tissues are carefully dissected away from the region of the graft. A patent graft is easily located by the persistence of the thrill which is found not only in the graft but in the distal portion of the coronary sinus as well.

Then the rolled-up Orlon sutures are located, sutured within the pericardium. They are carefully mobilized and are unrolled by dividing the

black arterial silk. The two Orlon sutures are separated and are then separately tied down over a 3 mm. proble which is laid on the coronary sinus. After tightly tying the suture, the sound is drawn out, thus providing a 3 mm. passageway through which the sinus blood can partly escape into the right atrium. This is repeated with the second Orlon suture (Fig. 14). The chest wound is then closed in the usual manner using a water-seal drainage system. This second stage operation is relatively short.

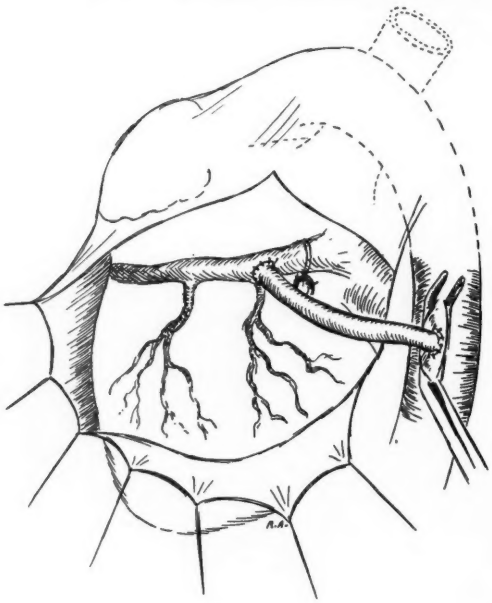

Fig. 13.

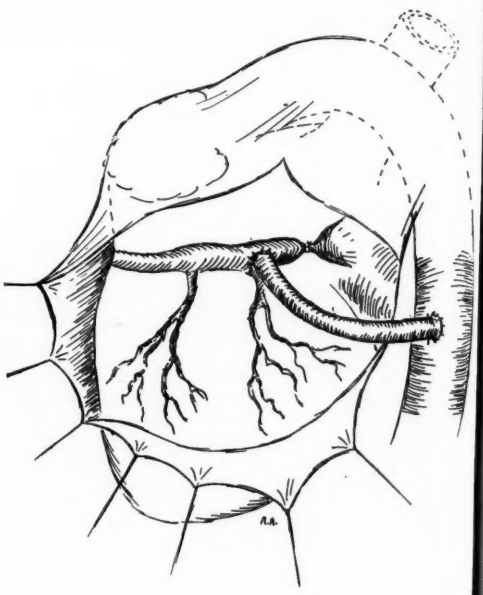

Fig. 14.

Fig. 13.—Completion of the aortic anastomosis and loosening of the aortic clamp for management of any possible leaks at the suture line which might be present.

Fig. 14.—Completed second stage, showing the partial occlusion of the coronary sinus by means of the encircling Orlon sutures.

TECHNICAL MODIFICATIONS

For theoretical and practical reasons (such as the incidence of thrombosis of the graft) we have recently suggested and carried out certain technical modifications of this operation. These have in no respect changed the underlying basic principles.

One of these has been an attempt to use the splenic artery rather than the aorta and a vein graft as the source of arterial blood. While this is technically possible in approximately 75 per cent of patients, it is unsatisfactory in about one-third of these because the terminal lumen of the splenic artery is too small. Thus only about one-half of all patients can expect a good arterialization of the coronary sinus by this means. Such an anastomosis was attempted in two of the present series. In one of them it proved to be impractical because of the small terminal size of the vessel.

Another technical modification, and a much more promising one, is the excision of a button of aortic wall rather than merely a longitudinal incision of the aorta (Fig. 15). Our attention was drawn to this detail when the observation was made during the first stage of one of these operations that the initial heavy flow through the graft became diminished and closed off within a period of fifteen minutes after it was established. This was apparently due to approximation of the lips of the aortic incision shutting off the flow. While this phenomenon has not been observed in experimental animals nor in children subjected to the Potts aortic-pulmonary anastomosis for tetralogy of Fallot, it is quite conceivable that it would not be uncommon in the middle-aged individual with an

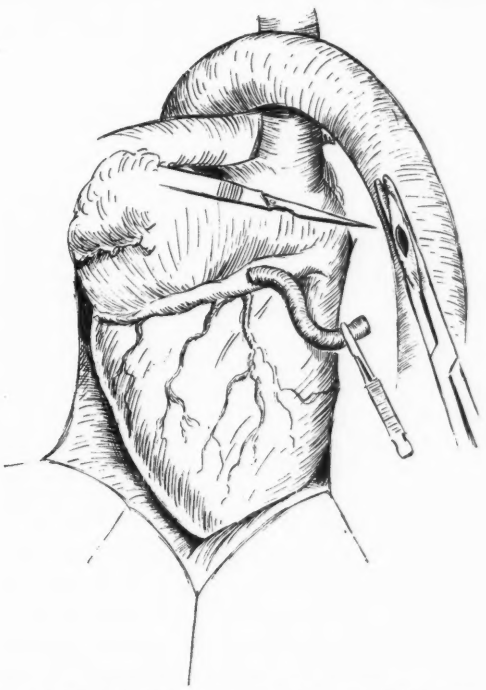

Fig. 15.—Preparation of the exteriorized segment of the aorta by means of resection of a "button" of aortic wall, rather than a linear incision.

arteriosclerotic aorta who is the usual candidate for arterialization of the coronary sinus. Such reduced or actually occluded flow in the graft would almost certainly lead to subsequent thrombosis. In two subsequent cases a button of aortic wall was removed. An unthrombosed graft was the result in each. Use of a free segment of the left subclavian artery, easily available at surgery, seems to provide a superior graft.

Another technical modification which we have carried out in one patient is the use of a continuous everting mattress suture for the aortic anastomosis. While both the vein graft and the coronary sinus have an inherent tendency

to eversion of the cut edge, thus rendering a simple continuous suture line very satisfactory for the anastomosis of vein to sinus, the cut aortic edge has no such tendency. Indeed, the aortic wall tends to separate into layers with the intima detached from the media. It is our feeling that an everting mattress suture would actually partially apply the vein graft into the aortic opening to seal off any such irregularities.

Finally, we have discontinued the practice of removing the adventitia from the aorta, since this provides a much stronger tissue for the insertion and holding of sutures. In an arteriosclerotic, perhaps excessively friable aorta, the considerable strength of the adventitia would seem well worth preserving. The old theory that retention of vascular adventitia predisposes to thrombosis of a vascular suture line was thoroughly disproved in a comparative experimental study by Jamison and Dugan.[18]

We feel that these modified technical details have been responsible for the somewhat improved results obtained with respect to the patency of the graft as observed at the second stage of the operation.

Recent discussion with Beck[19] regarding these technical modifications, particularly that of resection of a button of the aortic wall prior to performance of the anastomosis, has led to this wholehearted concurrence. In fact, he had early in his experience removed an aortic button in a human being. For some reason that technique was then abandoned, but will probably now be revived.

This report is based upon eighteen patients operated upon at the Hahnemann Hospital of Philadelphia and at the St. Michael's Hospital of Newark, N. J. The majority of these patients were operated upon by Dr. Claude Beck himself, and only the more recent of them have been independently operated upon by one of us (C. P. B.). We are endeavoring in this presentation to analyze the results obtained in this operative series and to indicate the various problems and pitfalls encountered.

Two patients in this series did not actually have an anastomosis performed. In one patient (W. C.) the coronary sinus was so delicate that it would not hold a suture for anastomosis, and was finally closed with difficulty. In the other patient (S. S.) the coronary sinus was found to lie too far to the right of the aorta, making anastomosis technically impossible.

There were two deaths in this series, a mortality of 11.1 per cent. One of these was due to sudden exsanguinating hemorrhage at the site of the aortic anastomosis on the seventh postoperative day. The aortic wall had seemed abnormal in this case at the time of surgery, and anticoagulant therapy (Dicumarol) had therefore been instituted and carried out during the entire postoperative period. Whether the abnormal aortic wall (site of an arteriosclerotic ulcer as seen at autopsy) or the anticoagulant therapy had been responsible for the secondary hemorrhage is debatable. This is the only patient in the entire series treated with anticoagulants. The graft was patent.

The other death occurred a few hours after surgery, apparently due to cardiac or circulatory depression. This patient had had two infarctions prior

to surgery, and presented a definitely enlarged heart. Today, we would exclude such a candidate from operative consideration.

One patient (W. S.) developed a pleural empyema after the first operative stage. Adequate drainage resulted in re-expansion of the lungs and complete obliteration of the pleural space. However, a second stage operation was not performed since this patient refused surgery.

In two patients (A. D. and N. R.) thrombosis of the anastomosis was disclosed at the time of the second operation. In one of these the splenic artery had been anastomosed to the sinus. This was treated by immediate application of a vein graft between the aorta and the coronary sinus. The other was treated by opening the graft and removing the clot. The Orlon sutures were then tied down in each case. No subsequent stages were ever performed. Both patients have manifested slight subjective improvement.

Of the remaining eleven patients, four were found at the second stage to have thrombosed grafts. They were treated by instillation of asbestos powder into the pericardium. One is slightly improved, two are unchanged, and one died six months after surgery of progressive disease.

The seven with patent grafts who had both operative stages have presented consistent evidences of improvement varying from what would seem to be "clinical cure" to moderate amelioration of symptoms.

It was our intention to present objective evidences of clinical improvement in this presentation.

Unfortunately, we find that this is a rather difficult undertaking. In the first place, the follow-up is very short for such a variable disease as coronary atherosclerosis. Our oldest patient is now only fifteen months postoperative.

Second, the only valid objective study method which we have employed throughout the entire series is serial electrocardiography. Unfortunately, ballistocardiography was not employed in the early cases. It is being applied in our later cases and will be extensively used in the future.

ECG.—The postoperative electrocardiographic changes observed thus far in human beings appear to resemble those seen in the experimental animals. These changes consist mainly of negative T waves in either the standard or unipolar leads. The full implication of these changes is not entirely understood. The period of observation has been too short to ascertain the degree and rate of return of these changes and their relation to the clinical state.

Consideration of the usual cardiologic methods of evaluating progress in patients with coronary artery disease reveals that they are all basically subjective.

Thus anginal pain, its severity, and its frequency is essentially subjective. Shortness of breath is also subjective unless it is associated with cyanosis, bronchospasm, or pulmonary edema. None of our patients presented any of these last clinical features.

Similarly, the so-called objective tests of work tolerance whether controlled (treadmill, Master's two-step test) or uncontrolled (climbing stairs,

walking, and working) are strictly limited by the former two features: angina and dyspnea, which halt continued activity.

Obviously radiography offered us nothing since the patients in this series did not have much myocardial enlargement and presented no pulmonary congestion, even preoperatively.

Since we excluded cases with congestive failure, we could not measure any diminution of liver engorgement nor of dependent edema.

Thus our evaluation was limited to essentially subjective evidences of improvement. By these criteria, all seven were considerably improved. Anginal pain was completely abolished in the six who had previously presented it. No nitroglycerin has been used by any of the patients postoperatively. Dyspnea was markedly reduced. Work tolerance was so increased that all have been able to return to their former occupations except one, a physician who is less than three weeks postoperative. Stair climbing ability has increased in each case.

All express a great sense of well-being with improvement in appetite and general outlook. Perhaps part of this euphoria is due to a sense of confidence that potential future coronary arterial occlusion will not produce myocardial infarction.

Our earliest case was a patient (J. C.) who had suffered three distinct myocardial infarctions. He has been converted from a cardiac invalid, who took 100 nitroglycerin tablets per week and was unable to walk fifty feet without pain, to a robust man who can with ease run at top speed completely around a city block.

Such important subjective evidence of improvement leads us to feel that a great fuctional change has taken place in each individual's cardiac action. More absolute indications must await other studies and the passage of much more time.

SUMMARY AND CONCLUSIONS

1. The concept that arterial blood can be delivered to the myocardial capillary bed by reversing the flow through the coronary sinus and its ramifications has been presented. Further anatomic and physiologic research is still necessary. Such studies are now under way (by some of us[16]).

2. These techniques show that the vascularity of the left ventricle and ventricular septum is much greater than that of the right ventricle, and that the venous system is far more abundant and extensive than the arterial.

3. After the Beck operation anastomotic communications between the epicardial veins of the left heart (ramifications of the coronary sinus) and the small cardiac and anterior cardiac veins of the right ventricle become markedly dilated. This suggests that they may offer an additional safety mechanism to permit escape of any excess blood entering the heart through the graft. Such collateral veins suggest the possibility of also improving the circulation of the right ventricle by the arterialization of the graft.

4. Existing physiologic information (Eckstein) bears out the probability of immediate reversal of flow through the superficial cardiac venous drainage after the second stage of the Beck procedure.

5. Both anatomic and physiologic evidence bears out the assumption that arterialization of the sinus in time brings about a progressive and great dilatation of the normally minute existing intercoronary communications. Extracardiac anastomoses between the coronary vessels and adjacent structures also become established and functioning. The relative amount of flow through these channels is still unknown.

6. The original Beck anastomotic technique is presented. Certain modifications have been suggested to lessen the incidence of technical failures. Chief among these is the removal of a button of aortic wall to produce a persistent rapid flow of blood through the graft and sinus. It is believed that thrombosis of the graft will, in the future, be a rarity because of this modification.

7. Criteria for the selection of suitable subjects for the procedure are suggested. Surgical survival figures in our experience indicate that such patients can easily tolerate the operation.

8. Objective evaluation of progress in cases of coronary disease under any form of treatment, medical or surgical, is both difficult and rare. The greatest clinicians manage these patients essentially with purely subjective guidance except for the information supplied by the electrocardiograph. Perhaps the ballistocardiograph or some other study mechanism may in the future offer us truly objective criteria.

9. The electrocardiogram is so changed by the reversal of blood flow through the coronary sinus in both dogs and human beings that it is valueless in measuring improvement or regression in these cases.

10. The theoretical advantages and the subjective improvement in patients who successfully undergo both stages of a Beck procedure are so great that we are encouraged to continue this operative program until time and improved methods of measurement ultimately establish its value.

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DISCUSSION

DR. CLAUDE S. BECK, Cleveland.—I should like to express my appreciation to Dr. Bailey and his group for the work they have done in this development. This operation has required assistance and I hope the work will receive the support of a good many of the surgeons in the audience.

I think I can say positively that the operation protects the heart against occlusion of a major coronary artery. It reduces the incidence of ventricular fibrillation after a major artery has been ligated, and it reduces the infarction or the destruction in the myocardium after the artery has been ligated. These, I believe, are statements of fact. We would like to know how to explain this benefit, and during the past year Dr. Eckstein and Dr. Leighninger have been doing experiments to show the mechanism by which this new circulation is beneficial to the heart. This physiologic study is difficult to carry out. They have been working on the study for one year, and it will require another year of full-time work to complete it.

Several conclusions can be drawn from the work so far. It has been shown first by Hahn in a few experiments, and during the past year by Eckstein and Leighninger and others working with them, that arterial retrograde flow from aorta, through graft and through capillary bed does occur. This blood does give up its oxygen. If the circumflex artery is ligated and then cut distal to the ligature, the blood comes out of the cut artery as venous blood. In order to give up its oxygen this blood from the graft must have gone through the capillary bed. Perhaps I should explain the type of experiment that was done. The graft of vein was placed between sinus and aorta. Three weeks later the sinus was partially ligated at its ostium in the right ventricle so that all the blood would not escape as in an arteriovenous fistula. At variable intervals thereafter the circumflex artery was dissected out, ligated at its origin, and cut distal to the ligature. The distal end was cannulated. Studies in back flow from the cut artery were made with the graft open and with the graft closed. I can only refer to these studies in general terms.

One, it has been shown that back flow through the capillary bed does occur. In the dog with normal coronary arteries it would appear that the quantity of such capillary back flow reaches a peak in several weeks and then tapers off and stops entirely. Whether this occurs in the human patient with coronary artery inflow disease we do not know. Two, it has been shown that the graft stimulates the development of intercoronary arterial anastomoses. Why it does this we do not know. In the normal dog's heart the amount of intercoronary back flow is about 2 to 4 c.c. per minute. That is, if the circumflex is ligated and cut the amount that escapes from the cut artery is only a few cubic centimeters and it comes from the descending coronary artery. It is red blood and it has not gone through the capillary bed. As time goes by and as actual retrograde flow from graft through capillary bed decreases, the intercoronary flow increases so that the retrograde flow may be as high as 22 c.c. per minute at the end of six months.

The third point that these experiments by Eckstein have shown is the importance of a few cubic centimeters of oxygenated blood to ischemic myocardium. As small an

amount as 5 c.c. per minute delivered to the muscle mass supplied by the circumflex artery, and this probably is half of the left ventricle, can prevent fibrillation and death. The importance of this does not seem to be generally realized. A fourth point is beginning to appear. They have done experiments in which the back flow from the severed artery was small and it was the same with the graft open or with the graft closed off. At the same time the electrocardiogram approached normal with the graft open and became abnormal with the graft closed off. Perhaps oxygen transfer can take place without the blood making a complete transit through the capillary bed and out the artery. The vascular bed on the venous side of the capillaries is extremely rich. Perhaps there is some ebb and flow here that can be beneficial without going through the capillaries.

It is fortunate that these studies are being made on the dog, and it is fortunate that we have as good a physiologist as Dr. Eckstein and as good a cardiac surgeon as Dr. Leighninger to do this work.*

There are no similar studies for the human heart. Severe coronary artery disease in the human heart produces circulatory conditions that cannot be duplicated in the dog heart. In the human heart with extensive and marked arterial occlusion one would expect the inflow to be greatly reduced and the capillary pressure to be greatly reduced. If these conditions exist then one would expect blood to flow backward more freely than in the normal heart. I have observed pinkening of vein and myocardium in the human heart with coronary disease after the graft has been placed and this pinkening has been marked.

So far I have performed this operation on sixty-two patients. The record is not entirely satisfactory. The one good feature about the record is that the operation is beneficial to patients with coronary artery disease. It is difficult to evaluate the status of coronary artery disease before and after operation. It is also difficult to make measurements of benefit on patients. How do we know whether the benefit that follows operation is due to the operation or would have occurred without the operation? This problem exists. I think I have seen enough good results to say that the operation is beneficial to patients with coronary artery disease. The mortality following operation depends upon the severity of the disease. The first twelve patients operated upon were totally or almost totally incapacitated. Eight of these patients died. Then we took a better type of patient and there was one death among 19 patients. Excluding the first twelve cases the mortality is 26 per cent. I think we could reduce mortality to about 10 per cent if we selected patients more carefully. However, it is the disease rather than the operation that contributes to mortality. Some of our patients died before the operation was done. In some of the patients the graft became thrombosed. In some the aorta was severely diseased so that the graft was not placed. I think we might be able to overcome the problem of thrombosis by cutting out a small piece of aorta as Dr. Bailey suggested. We used to do this in our early work but gave it up because it was not necessary on dogs and often the opening into the aorta became too large.

In conclusion, I should like to suggest that some of you surgeons become interested in this development. After some preliminary training on dogs the operation can be done satisfactorily on patients. The experimental work shows that the operation is beneficial. However, the final answer will come from the patients and will be decided upon through results as follows: (1) relief of pain, (2) ability to work, and (3) duration of life after operation. So far no patient who left the hospital with a patent graft has died.

DR. ARTHUR VINEBERG, Montreal.—I would like to take this opportunity to congratulate Dr. Bailey on his excellent presentation. I feel that the Beck method of revascularization of the heart seems to have possibilities and may even become the procedure of choice. It has, however, certain disadvantages and, for that reason, I should like to draw to your attention another method of revascularization of the myocardium, which I have presented before this Association on two separate occasions.

The method I refer to is internal mammary artery implant. You will recall that the left internal mammary artery is detached from the chest wall and placed within a tunnel in the

*This work is supported by a grant from the United States Public Health Service.

ventricular myocardium. We have shown in the past that this artery forms new branches which join with the left coronary circulation.

(Slide.) This shows an implanted internal mammary artery injected a few months after its implantation into the left ventricular myocardium. The injection mass was a rubber compound resisting acid digestion. The injection mass entered the internal mammary artery, was seen to spread through the anterior descending arteries of the left coronary, and come out of the anterior descending artery. A piece of polyethylene tubing was tied around the origin of the anterior descending branch left coronary and the whole specimen digested. The cast shown in this slide shows quite clearly an intimate junction between new branches of the internal mammary artery and branches of the anterior descending branch of the left coronary artery.

In all our injection specimens, the injection mass has entered the internal mammary artery and come out of a cannula placed in the left coronary artery, leaving little doubt that there is a true mammary coronary anastomosis.

(Slide.) It has been suggested by some that the anastomosis is superficial; this slide and the subsequent slide show the injection mass distributed through the entire left ventricular myocardium and from the inner to the outer surfaces of the heart.

It has also been suggested by some workers that the branches that are formed by the internal mammary artery are not truly arterial in character, but are capillaries which tend to disappear in a few weeks. This slide shows that the anastomosis is present up to fifty-eight weeks.

(Slide.) This slide shows an internal mammary artery which has been in the heart of a dog for over ninety days and represents a study made by the serial section of the anastomosed vessels with special stains for elastic tissue and muscle; note the elastic and muscle elements in one of the newly formed branches of the implanted internal mammary arteries. Serial section of seven different anastomoses has been thus studied with similar results.

Anatomic proof of a mammary coronary anastomosis is insufficient; for this reason functional experiments have been conducted to ascertain the value of the anastomosis. The first of these was that of survival studies following that of ligation of the left coronary artery.

(Slide.) A comparison is made between some of the figures after arterialization of the coronary sinus (Beck) and after internal mammary artery implant. It will be noted that from Beck's figures in the control series, there was a 70 per cent mortality rate with a 30 per cent survival with infarction. Whereas, after arterialization of the coronary sinus, there was a 10 per cent mortality and a 45 per cent survival with infarction and only 45 per cent survival without infarction.

In our own series, our control figures are approximately the same as those of Beck, namely, 80 per cent mortality and 20 per cent survival with infarction after internal descending branch ligation. Whereas in twenty-nine animals where there was a good mammary-coronary anastomosis, there were no deaths and all animals survived without infarction.

Others have published a much lower mortality rate for anterior descending branch ligation. The discrepancy between these figures and those obtained by Beck and ourselves I believe can be explained. The mortality rate is dependent on the number of branches tied, therefore it is necessary to be certain that the ligature placed around the anterior descending branch of the left coronary artery is located at its origin.

At autopsy, a careful search must be made for other branches arising from the left coronary artery itself which have not been included in the ligature and which could maintain life. A second experiment indicating the value of mammary-coronary anastomosis is shown on the following slides:

(Slide.) This animal survived an anterior descending branch ligation and six weeks later the internal mammary artery was tied off; the dog died forty-eight hours later with a large anterior wall infarct, indicating pretty clearly that the blood supply to this ventricle was largely supplied by the implanted internal mammary artery.

I am simply recalling this procedure to your attention. We have now operated on seven patients; there have been two deaths; three of the surviving patients are free of pain and are working; a fourth has been done recently. There has been one failure to date.

DR. HARRISON BLACK, Boston.—There are a few comments I would like to make on this paper. One point that should be borne in mind in the evaluation of any procedure for coronary insufficiency is that pain, although the most dramatic, is yet a poor criterion by which to judge the success of the operation. Many different events in our experience have resulted in the relief of angina without detectable prolongation of life. Among these are a dorsal sympathectomy, scarification of the epicardium, a myocardial infarction, or a period of shock. For this reason I believe we will have a better answer if we judge the various methods on the basis of survival. This admittedly is going to take a considerable period of time.

So far as experimental work is concerned, it seems to me that the crux of the problem is whether the procedure produces an increased blood flow in the capillary bed and thereby better oxygenation of the myocardium. This remains to be demonstrated conclusively for any of the current techniques and the method of Goldman mentioned earlier in this meeting may find application here. It is also worth noting that Palmer and Welch* have demonstrated that reversal of circulation in the lower extremity by femoral artery to femoral vein anastomosis is followed by deficient peripheral circulation, and they conclude that the end result differs little from a conventional arteriovenous fistula. The reason for this is that the blood is shunted through venous collaterals back to the heart, by-passing the capillary bed. Whether a similar situation obtains in the reversal of circulation procedures which have been described today remains to be seen. However, I think it is a possibility that must be seriously considered.

Another point to remember in evaluating experimental results is that in the dog survival rates following anterior descending coronary ligation can be improved by a variety of operative maneuvers in addition to the ones mentioned today. Furthermore, Schlesinger has been able to demonstrate by his injection techniques in human beings that a hemoglobin of less than 70 per cent for more than two months will produce intercoronary communications in more than 70 per cent of persons. It therefore must be proved that any procedure in question accomplishes more than these nonspecific stimuli.

I would like to mention an additional technique which we have used in the experimental laboratory and on two clinical cases. We have not felt that the approach introduced by Dr. Beck of producing vascular adhesions to the heart has been entirely exhausted. One of the observations we have made—and this is borne out by the work of Burchell at the Mayo Clinic—is that the epicardium is the barrier to the ingrowth of these vessels. We have found in the laboratory that if the epicardium is removed, however, vascularization of the myocardium is much improved. We have tried many methods of removing the epicardium using sharp dissection, bone dust, asbestos, trichloracetic acid, talc, aluronate paste, iodine, chlorine, etc. Only when 95 per cent phenol was used were we able to de-epicardialize the ventricle effectively without traumatizing the heart and thus obtain ingrowth of vessels into the myocardium which are large enough to be penetrated by the Schlesinger mass (lead phosphate in agar).

(Slide.) This slide shows a heart which has been painted with 95 per cent phenol. You can see the marked degeneration of the epicardium while the myocardium remains unaffected.

(Slide.) On the right are vascular adhesions which have been established following removal of the epicardium. On the left, new vessels have appeared in the myocardium and these are large enough to take the Schlesinger mass.

(Slide.) This is also a dog experiment, showing injection of the agar mass via the aorta as in the previous slide, with the proximal aorta clamped off so that the mass cannot

*Surg., Gynec. & Obst. 94: 206, 1952.

enter through the proximal coronary circulation. Above we see the bronchial arterial branches injected in the lungs, and in the heart itself there are vascular channels which are filled with the mass.

DR. BAILEY (Closing).—We have been very much interested in the work of Dr. Vineberg, have attempted to repeat his experiments in the laboratory, and have found some interesting corroborative evidence. However, we have not done enough yet to come out unequivocally and say that we have completely repeated them.

There is no doubt, as Vineberg has shown, that it is possible to place a large bleeding vessel into the myocardium and not get a hematoma. There is no other soft tissue that I know of that can offer that advantage to the surgeon. I think that observation in a sense begins to answer Dr. Black's comment about other organs in which reversal of circulation has not worked. The heart is of a peculiar spongelike structure, with big myocardial sinusoids and a rich capillary bed. It can drain off large quantities of blood and this drainage can compete with actively bleeding vessels.

It is true, as Dr. Black has pointed out, that pain is a poor criterion for the measurement of improvement in cases with coronary artery disease. So is dyspnea, because that also is a subjective symptom. Yet I note that all the eminent cardiologists who treat coronary artery disease do so on the basis only of subjective symptoms, except for the electrocardiogram. The ballistocardiogram may change that situation in the future. But at present all work tolerance tests are sharply limited by the onset of pain and dyspnea; so again they are actually subjective tests. I question them because we have been unable to evaluate these cases that way. Unless we hope to be better than the cardiologists themselves, we will have to accept subjective improvement as a therapeutic guide.

Those patients who had grafts which became thrombosed might be considered, in a sense, as controls to measure the effect of pericardial poudrage. There should have been improvement of pain. We put asbestos in the pericardium and certainly produced lots of pericardial adhesions. One of the patients does not have pain. All the rest do. They do not have the great improvement subjectively that the patients with open grafts present.

As to the test as to whether the myocardium actually does get better oxygenation, that is going to be quite a task and we have not figured out how that is going to be done.

The epicardium is, as Dr. Black has pointed out, a barrier to the formation of capillaries from pericardial adhesions. As you all know, Dr. Beck used to use a burr to take off the epicardium for that reason in his myopexy operations. He also used 90 per cent phenol, or perhaps it was 100 per cent, and twenty-seven other substances in the attempts to produce vascularized pericardial adhesions, in an experiment that was reported many years ago. Of them all, he felt that asbestos was by far the best available substance because it not only produced vascularized adhesions but also stimulated the development of the intercoronary communications.

I might say something about the electrocardiogram after arterialization of the sinus. The electrocardiogram in dogs and in human beings shows changes which might be interpreted as indicating a worsening of the condition. Since the dogs do not have coronary disease we do not think these changes have their usual significance. Therefore we believe that the one objective method cardiologists use for measurement of improvement in this disease is worthless for our purposes, because the electrocardiogram is apparently so greatly changed when the blood flow is reversed through the coronary sinus and its tributaries.

PULMONARY VALVULOTOMY

Description of a New Operative Approach With Comments About Diagnostic Characteristics of Pulmonic Vai vular Stenosis

Harris B. Shumacker, Jr., M.D., and Paul R. Lurie, M.D. (by invitation)
Indianapolis, Ind.

SINCE the first successful pulmonary valvulotomies were performed by Brock[1] and Sellors[2] in 1948, this procedure has found increasing applicability in the treatment of congenital heart disease associated with stenosis of the pulmonic valve. Thus far, however, the total reported experiences with this method of treatment are relatively few. It is the purpose of this communication to describe a new operative approach, to make some comments about the diagnostic characteristics of pulmonic stenosis not associated with ventricular septal defect, and to present the results of treatment in a small series of cases.

THE OPERATIVE APPROACH

The operative procedure described by Brock[1, 3, 4] has proved generally satisfactory, and others[5-7] have followed it in essential detail. Use of the left anterior intercostal approach, however, has one potential disadvantage—the difficulty in certain cases of dislocating the heart into the left thorax so that traction sutures can be placed in the anterior wall of the right ventricle preliminary to cardiotomy and valvulotomy. This is particularly true in the less severe cases without marked right ventricular enlargement. Such difficulty was encountered in four of the first six valvulotomies performed through a left anterior thoracotomy. Indeed, in two of them, efforts at manual displacement of the heart into the left thorax were so regularly attended by alarming changes in cardiac rate and rhythm that it proved necessary first to place a suture in the wall of the presenting left ventricle and, by traction upon it, to expose the anterior right ventricular wall. As a consequence of these experiences the use of Milton's midline sternal-splitting approach was investigated. It was found to give excellent exposure and has been used with great satisfaction in the last three patients in whom a clear-cut preoperative diagnosis of pulmonic stenosis could be made. It has also been used in two additional patients who proved to have truncus arteriosus.*

The operation is performed in the following manner: The chest is prepared widely in the event the surgeon might wish to extend the incision into either

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Aided in part by a grant from the James Whitcomb Riley Memorial Association.

Read at the Thirty-second Annual Meeting of The American Association for Thoracic Surgery at Dallas, Texas, May 8-10, 1952.

*In one, pulmonic stenosis was considered a possibility because of known right ventricular hypertension without evidence of left to right shunt and inability to pass a catheter into the pulmonary artery. The other was thought to have tetralogy of Fallot. A side-to-end aortic pulmonary artery shunt using the left subclavian artery as a free graft had brought about considerable but incomplete improvement. An effort to perform a valvulotomy or infundibulectomy was considered safer than an attempt to produce an additional systemic-pulmonary artery shunt in view of the fact that one pulmonary artery had been divided and one subclavian sacrificed.

side of the thorax. An incision is made in the midline from the cephalad end of the manubrium to the lower end of the xiphoid. The incision is extended down through the periosteum which is stripped back from the midline a few millimeters on either side. The superior and posterior aspects of the upper end of the manubrium are freed sufficiently to permit engagement of the Lebsche knife. The sternum is split through its entire length. Any bleeding of the cut edges is controlled with bone wax or Oxycel or Gelfoam. The two halves of the sternum are retracted widely with one or two Rienhoff rib spreaders. A few cubic centimeters of 4 per cent procaine solution are injected into the pericardial sac. The lower portion of the thymus is dissected from the pericardium and reflected upward. The pericardium is incised in the midline after placing traction sutures in it, and the cut edges are retracted. Excellent exposure is obtained (Fig. 1). The pulmonary artery and the adjacent portion of the right ventricular wall are more or less in the middle of the field.

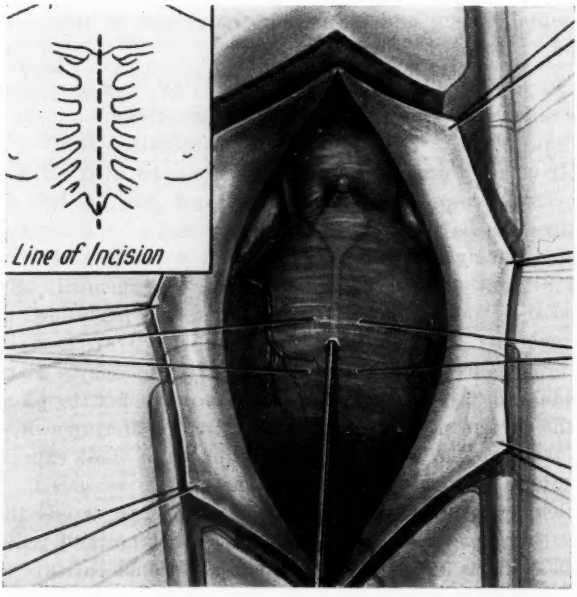

Fig. 1.—Drawing of the operative exposure.

The operation then proceeds according to the usual method. Procaine is injected into the ventricular wall at the point selected for cardiotomy. Traction sutures of silk are placed and a small stab wound is made. Exploration of the ventricle and pulmonary artery is carried out by passage of a small Hegar dilator. Either the Brock or the Potts valvulotomes are used for cutting the stenotic valve. Dilatation is carried out with the Potts dilator or by spreading a Kelly clamp. Bleeding from the ventricle is controlled by digital compression and by crossing of the traction sutures.

After completion of the dilatation the cardiotomy incision is repaired with a few interrupted silk sutures. The pericardial cavity is irrigated with normal saline solution and the lower portion of the pericardial incision is closed loosely with a few sutures of silk. That portion overlying the pulmonary artery is left open for drainage. Small Penrose drains are brought out through the upper and lower ends of the wound. If the pleural cavity has been inadvertently entered, the opening is enlarged and a catheter is introduced through an interspace. The sternum is approximated with a few sutures of stainless steel wire. In small children the sternum is soft and the sutures may be placed with a heavy cutting-edge needle. In older individuals they are passed through small drill holes. The periosteum, the deep and superficial fascia, and the skin are closed carefully with interrupted silk sutures. Upon return to the ward the patient is placed in an oxygen tent or is given oxygen through a nasal catheter. He is encouraged to lie in the prone position for the first few days in order to provide dependent drainage of the pericardium.

It is apparent that this approach is best utilized in cases in which preoperative studies point clearly to the diagnosis of pulmonic stenosis. Should the need arise it would seem feasible to extend the incision into either pleural cavity by transecting the appropriate half of the sternum and making an intercostal incision. Obviously in cases in which transthoracic exploration is felt to be of primary importance the sternal-splitting incision should not be used.

PULMONIC STENOSIS WITH INTACT VENTRICULAR SEPTUM

Pulmonary valvulotomy was performed in eight patients thought to have pulmonary valvular stenosis without ventricular septal defect. One patient was severely incapacitated. The others had mild to moderate disability (Table I). All had some dyspnea on exertion but only in two was it severe. All had fatigability, but it was extreme in only one. One patient had marked and five had only mild cyanosis. Cyanosis was not clinically evident in one patient and was only questionably present in another. Clubbing was extreme in one patient and mild in three; no definite clubbing was observed in four. All had a systolic murmur in the pulmonic area. A thrill was readily palpable in seven and suggestively present in the eighth case. In none was there a pulsating liver. The blood pressure tended to be normal or low. In Cases 2, 4, and 8 the systolic pressure ranged between 90 and 94. Hemoconcentration was very marked in one patient. Two had evidence of slight hemoconcentration, and there was no hemoconcentration in the remainder.

There was electrocardiographic evidence suggesting right ventricular enlargement in all except one case, but there was clear-cut evidence of right ventricular enlargement by fluoroscopic and radiologic study in only three and questionable evidence in two others. In three patients there was nothing to suggest specific chamber enlargement. In all except one there was enlargement of some portion of the pulmonary artery. In most of them the pulmonary trunk appeared dilated (Fig. 2). In several, however, there was no convexity in the cardiac segment representing the region of the pulmonary trunk but rather dilatation of the left or both main pulmonary arteries (Fig. 3). It was

TABLE I. SYMPTOMS AND SIGNS IN PATIENTS WITH PULMONIC VALVULAR STENOSIS AND INTACT VENTRICULAR SYSTEM

CASE NO.	AGE	SEX	DYSPNEA*	FATIGABILITY*	CYANOSIS*	CLUBBING*	R.B.C.	HEMATOCRIT	EVIDENCE OF RT. VENTRICULAR HYPERTROPHY		X-RAY EVIDENCE OF ENLARGED PULMONARY ARTERY	HIGH P WAVES	PERIOD OF FOLLOW-UP IN MONTHS
									X-RAY	ECG			
1	17	M	++	++	+	+	5.96	55	0	+	+	0	13
2	9	M	++	++	+	0	4.48	39	±	+	+	0	10
3	11	M	+	++	+	0	5.96	—	0	+	+	0	8
4	10	M	++++	++++	++++	++++	9.90	83	+	+	0	+	8
5	11	F	++	++	+	+	4.03	40	+	+	+	+	7
6	10	M	++	++	±	0	—	42	0	0	+	0	6
7	25	F	++	++	+	0	4.39	—	+	+	+	0	6
8	6	F	+++	++	0	±	4.10	—	±	+	+	0	2

*Listed according to severity from + to ++++; + indicating very mild, ++++ very severe.

felt by some observers that these pulmonary arteries were perhaps more translucent than would be expected in normal vessels of the same size. No intrinsic pulsation was noted in them fluoroscopically. One patient had a markedly enlarged heart. The others were either normal in over-all size or only slightly enlarged. The P waves were high in the electrocardiograms of only two of the eight patients.

Arterial oxygen saturation was determined in all (Table II) and cardiac catheterization* (Table III) in all save one. The exceptional patient was a diminutive boy of 10 (Case 4) who was extremely ill. He had marked cyanosis,

Fig. 2.—Roentgenograms of Cases 2 (*A*) and 8 (*B*) showing dilatation of the pulmonary trunk.

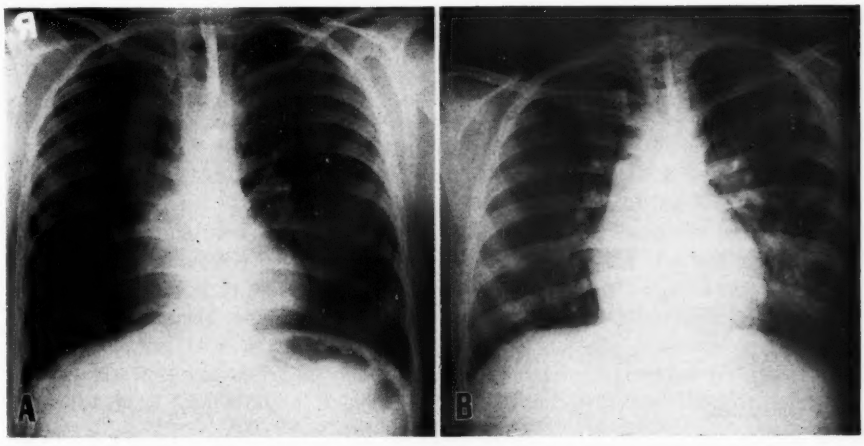

Fig. 3.—Roentgenograms showing dilatation of pulmonary arteries in Cases 3 (*A*) and 5 (*B*). In Case 3 it is clear that the region of the main pulmonary trunk is not enlarged. The left pulmonary artery is dilated, however. In Case 5 the main pulmonary trunk segment seems diminished in size but there is obvious dilatation of its main branches.

*The technique used is described in detail elsewhere.[5]

TABLE II. ARTERIAL OXYGEN SATURATION STUDIES IN CASES OF PULMONIC STENOSIS WITHOUT EVIDENCE OF VENTRICULAR SEPTAL DEFECT

CASE NO.	TIME OF TEST	BREATHING ROOM AIR	BREATHING OXYGEN			CLASSIFICATION
			LOW	MEAN	HIGH	
1	Preoperative	89.0	89.2	90.9	92.6	Definite veno-arterial shunt
2	Preoperative	91.2	95.9	98.1	100.3	Borderline, possible small veno-arterial shunt
	Postoperative (8.5 mo.)	92.2	97.2	99.3	101.4	Borderline, possible small veno-arterial shunt
3	Preoperative	91.4	94.4	96.5	98.6	Definite veno-arterial shunt
4	Preoperative	56.9	61.9	63.2	64.5	Definite veno-arterial shunt
	Postoperative (3.5 mo.)	90.2	88.2	89.0	90.5	Definite veno-arterial shunt
5	Preoperative (1)	91.3	94.6	96.6	98.7	Definite veno-arterial shunt
	Preoperative (2)					
	Resting	89.3				Definite shunt
	Standing 5 min.	94.5				No shunt
	Exercise 1 min.	88.9				Definite shunt
	After exercise	96.8				No shunt
6	Preoperative	87.7	86.3	88.5	90.6	Definite veno-arterial shunt
	Postoperative (6.5 mo.)					
	Resting	94.3	98.1	100.5	102.9	Borderline, possible small veno-arterial shunt
	Exercise	92.6				
7	Preoperative	90.7	98.7	100.9	103.2	Definite lung factor, probably no veno-arterial shunt
	Postoperative (4 mo.)					
	Resting	91.7	100.8	103.1	105.5	Borderline, probable lung factor
	Exercise					Normal, probably due to exercise hyperventilation
8	Preoperative	91.1	94.3	96.7	99.1	Definite veno-arterial shunt

TABLE III. PRESSURE STUDIES IN CASES OF PULMONIC STENOSIS WITHOUT VENTRICULAR SEPTAL DEFECT

CASE NO.	PREOPERATIVE					POSTOPERATIVE				PERCENTAGE FALL IN R.V. SYSTOLIC PRESSURE AFTER OPERATION
	R.A.	L.A.	R.V.	P.A., AT VALVE	P.A., DISTAL	R.A.	R.V.	P.A., AT VALVE	P.A., DISTAL	
1	9/1	5/−1	120/0	2/−2	2/−2					
2	—	—	65/−7	10/−1	16/4	—	39/−5	12/2	18/6	40
3	16/2	21/−3	82/−6	24/6	46/5					
5	0/−6	—	105/9	25/16	46/13					
6*	3/−2	—	57/0	11/1	19/3	9/5	48/6	20/8	20/9	16
7	1/−4	—	127/−7	1/0	5/8	4/−6	84/−3	14/4		34
8	2/−5	—	142/2	17/2	17/5					
By ventricular puncture										
Before valvulotomy			90/0							
After valvulotomy			64/0							

*Patient not in basal condition during postoperative study.

clubbing, hemoconcentration, and a large heart (Fig. 4). The liver was enlarged, heart sounds were faint, and he had a gallop rhythm. In this patient the resting arterial oxygen saturation was only 56.9 per cent. The remainder had only mild or questionable evidence of arterial oxygen unsaturation, the values ranging from 87.7 to 91.9 per cent. From these observations and values obtained during the breathing of pure oxygen, a definite right to left veno-arterial shunt can be presumed to be present in six and a possible small shunt in one additional patient (Case 2). In Case 7 it is clearly established that the arterial hypoxia was due to a lung factor and there was no definite evidence of veno-arterial shunt. None of the usual explanations for increased lung factor in congenital cardiac conditions were applicable in this case. In two of those with definite right to left shunt (Cases 1 and 3) the cardiac catheter passed readily into the left atrium and the right atrial pressure was elevated; it is

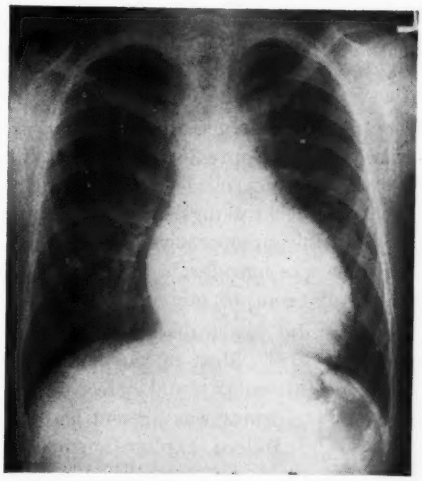

Fig. 4.—Roentgenogram in Case 4. The heart is seen to be greatly increased in size. There is evidence of right ventricular enlargement. The pulmonary artery segment does not appear remarkable.

entirely likely that a true atrial septal defect rather than a patent foramen ovale was present. That intermittent arterial hypoxia resulting from the intermittent shunting of blood from right to left atrium may occur in some is illustrated by studies carried out in Case 5. On one occasion the resting arterial oxygen saturation in this patient was 89.3 per cent. After standing for five minutes it was 94.5 per cent. At the end of one minute's exercise it was 88.9, and afterward 96.8 per cent. In none of the patients subjected to cardiac catheterization was there any evidence of a ventricular septal defect with left to right shunt from study of oxygen content of right atrial and right ventricular blood.

Pressure studies* made at the time of cardiac catheterization, and in one case by direct ventricular puncture at the time of operation, were of considerable interest. The right ventricular pressures ranged between 57 and 142 mm. of

*Made with the Hathaway manometer.

mercury. In one instance, at the upper extreme (Case 8), the systemic systolic pressure was exceeded by 40 mm. Right ventricular pressure was found to be near the systemic level in three patients (Cases 1, 5, and 7). In the other three patients the ventricular pressure was lower and was in the range in which as yet little experience with pulmonary valvulotomy has been obtained.

All of the pressures measured just beyond the pulmonary valve were lower than those measured more distally. This fact may be due to locally increased rate of flow with conversion of potential to kinetic energy at this point, followed by reconversion to potential energy more distally, in accordance with Bernouilli's principle. In five of the patients the pressure of the pulmonary artery near the valve was subnormal. In the two in which it was normal just beyond the valve, it was actually elevated more distally (Cases 3 and 5). The right atrial pressures were elevated in only two cases (Cases 1 and 3), in both of which entry into the left atrium was possible. The pressures in the right exceeded those in the left atrium during at least part of the cardiac cycle in these two cases. There was no frank right ventricular failure in any of the patients as evidenced by the normality of the ventricular diastolic pressures.

The single study by needle puncture of ventricular pressure at the time of operation revealed a fall in systolic pressure of about 30 per cent after valvulotomy. It will be interesting to learn whether the ultimate fall in ventricular pressure is predictable from such tracings made at the time of operation with the pericardium open. During preoperative catheterization the systolic ventricular pressure in this case was considerably higher than at the time of operation, 142 as compared with 90 mm. of mercury.

The diagnosis of congenital heart disease had been made early in life, usually before the age of 4 years. Most of the patients had apparently been thought to have an atrial or ventricular septal defect. The one patient in whom a diastolic as well as a systolic murmur was present had been considered as having a patent ductus arteriosus. Indeed, exploration had been carried out some months before the correct diagnosis was established.

During operation the pulmonary artery was found to be moderately or markedly dilated in all except one patient. In these cases the stenotic valve could be palpated through the pulmonary artery and a thrill could be felt with each systole. In the exceptional patient (Case 4) the pulmonary artery was small, having a diameter of less than a centimeter, and no thrill could be felt. The valvular stenosis in this, as in all the other cases, could be confirmed by palpation with probe and valvulotome, and the pulmonary artery began to pulsate vigorously immediately after valvulotomy and expanded to almost twice its former diameter. In one patient (Case 8) the valve was palpably calcified.

All of the patients have done well since operation. They have increased exercise capacity and, in general, are essentially free of symptoms within the limits of activity thus far permitted. When cyanosis was present beforehand it has disappeared or is milder in degree and less frequent in occurrence. The change in status following valvulotomy can be illustrated by those with the mildest (Case 3) and the severest (Case 4) symptoms.

The former patient was thought by his parents to exhibit dyspnea only rarely and after undue activity. Only infrequently was cyanosis of the lips noted, generally during periods of excitement. Though he could walk slowly for fairly long distances he tired easily and was always found to be sitting down to rest shortly after engaging in any play. Now eight months after operation he seems entirely normal. He plays vigorously, rides a bicycle well, and does not seem to tire at all.

The latter patient was so limited in exercise tolerance he could walk only a few steps at a time. He was markedly cyanotic and was forced to lead the exceedingly restricted life of a cardiac cripple. Because of poor heart sounds, gallop rhythm, and large liver, he was considered on the verge of heart failure and was digitalized before operation. From the moment of valvulotomy his color was excellent. He was discharged from the hospital on digitalis and with strict limitation of activity. About one week later he was readmitted in frank failure with marked edema probably resulting from excessive activity and too high a salt intake. He responded promptly to bed rest, salt restriction, and mercurial diuretics. Digitalis was discontinued a little less than three months after operation. Three and one-half months after valvulotomy the arterial oxygen saturation had risen from 56.9 to 90.2. It did not fall with mild sustained exercise (20 climbs up and down steps during a period of 2 minutes). The erythrocyte count had fallen from 9.90 to 4.43 million, the hematocrit from 83 to 52. He had gained from fifty-one to fifty-eight pounds and had grown in height. He now walks vigorously and has noted no fatigue on walking several blocks. His activity is being rapidly expanded.

In three additional patients postoperative arterial oxygen studies have been made (Cases 2, 6, and 7). All had higher values during breathing of air and during breathing of oxygen. Preoperatively one (Case 2) had a borderline condition with possible small veno-arterial shunt. The postoperative findings, though improved, were still in the same borderline classification. The second patient (Case 6) had evidence of definite veno-arterial shunt before operation. After operation the studies indicated a borderline situation with possible small shunt. The third patient (Case 7) had arterial hypoxia due to a lung factor before operation. Postoperatively she showed definite improvement but still had to be classified at rest as borderline with probably lung factor. During exercise after operation, probably as the result of hyperventilation and consequent increase in alveolar oxygen tension, the oxygen saturation rose to a definitely normal value. On cardiac catheterization these same three patients showed a reduction in right ventricular pressure, though not to normal levels, and a corresponding increase in pulmonary artery pressure.

It is of interest that a good result was obtained in the two patients in whom preoperative studies had demonstrated normal pressure values just beyond the pulmonary values and actually elevated pressures more distally. The outcome in one (Case 3) has been described. The other (Case 5) was digitalized during the postoperative period because of tachycardia. She has now been without digitalis for seven weeks and it is certain that she is considerably improved over

her preoperative state. The demonstration of pulmonary hypertension in two patients with pulmonic valvular stensosis is a surprising and, at present, unexplained occurrence. Our experiences demonstrate that, at least in mild degrees, it does not constitute a clear contraindication to valvulotomy.

OTHER PATIENTS TREATED BY VALVULOTOMY OR DILATATION OF THE PULMONARY ARTERY

Two additional patients have been treated by pulmonary valvulotomy and another by dilatation of a calcific stenosis of the pulmonary artery. One was a 21-year-old man thought to have tetralogy of Fallot. He had been severely incapacitated. Ten months earlier a left thoracotomy had been performed in the hope of establishing an aortic-pulmonary or subclavian-pulmonary shunt. The mediastinal area and the hilum of the lung were filled with a mass of wormlike collateral channels. In spite of care, bleeding was so troublesome that, alone in our experience with tetralogy of Fallot, it was felt necessary to abandon the attempt to isolate the pulmonary artery. A month later a segment of azygos vein was interposed as a free graft between the proximal ends of the divided subclavian artery and the upper lobe branch of the pulmonary artery. Though he was somewhat improved symptomatically and had less severe hemoconcentration, he was still badly handicapped and a continuous murmur was never clearly audible. Ten months after the first operation another exploration was made through the left thorax. At this time the hematocrit was 63. It had been 77 before the previous operations. Again the pulmonary artery could not be safely isolated. The lateral incision was extended anteriorly and a calcific stenosis of the pulmonary valve was discovered. Valvulotomy was carried out with good result. During the eleven months since operation he has been relatively well and has been working regularly. His color is excellent and the erythrocyte count normal.

A second patient was a pitifully handicapped 14-year-old girl with marked cyanosis, clubbing, hemoconcentration, physical incapacitation, and lethargy. From careful study including angiocardiography she was thought to have atypical tetralogy of Fallot with marked overriding of the aorta and unusually small pulmonary arteries. A left lateral thoracotomy was made. Preliminary dissection of the hilar area convinced the operator that if a pulmonary artery were present, it was diminutive in size. The incision was extended anteriorly and the pericardium opened. The main pulmonary artery was about normal in size or a little smaller than normal. The valve was not palpable. About 2 cm. distal to its origin, at about the region of the bifurcation, the artery became very small and there was obviously a calcified stenotic lesion in this area. A cardiotomy was performed and the calcific ring thoroughly dilated by spreading a Kelly clamp. Following this procedure, good pulsation was evident in the small left main pulmonary artery and a thrill could then be felt. The patient has been remarkably improved. On her first return visit three weeks after operation her color was good. She was much more energetic than she had been and could go up and down a flight of steps without dyspnea. Her activity is being rapidly increased.

A third patient was an 8-year-old boy with atypical tetralogy of Fallot which functioned essentially as a case of pulmonic valvular stenosis without ventricular septal defect. He had moderate dyspnea, fatigability, cyanosis, and clubbing but no hemoconcentration. The fluoroscopic and radiologic examination revealed right ventricular enlargement and decreased pulmonary artery shadows (Fig. 5). Angiocardiograms performed with anteroposterior and left anterior oblique projections revealed fairly large pulmonary arteries, suggestive evidence of infundibular stenosis, and normal sequential filling of the right and left sides of the heart, though some observers thought there was faint opacification of the aorta in one film at the time of right ventricular opacification. Cardiac catheterization studies revealed no increase in oxygen content in the right ventricle over that of the right atrium. A bubble in the system caused a slight damping effect with artificial elevation of diastolic, and lowering of systolic, pressures. Under the circumstances the right ventricular pressure was 67/9 and pulmonary artery pressure 15/6. Arterial oxygen saturation was 88. The patient was

explored for pulmonary valvulotomy. There was no visible abnormality in the region of the infundibular chamber. A jetlike systolic thrill was palpable in the pulmonary artery. No clear-cut stenosis was, however, palpable nor could one be sure of any infundibular stenosis. The Brock valvulotomes were passed and the pulmonary artery and infundibular chamber thoroughly dilated. The patient was considerably improved. He noted no dyspnea postoperatively, and his capacity for exercise was greatly improved. About thirteen months after operation he became acutely ill with a cold followed shortly by marked respiratory difficulty. He died within a few days. Post-mortem examination revealed the cause of death to have been massive left atelectasis from aspiration of foreign material which plugged the smaller bronchi. The heart showed right ventricular enlargement, a small high ventricular septal defect about 0.5 cm. in diameter with an overriding aorta, a moderate stenosis of the short infundibular chamber and the origin of the pulmonary artery, a large atrial septal defect, and drainage of the right pulmonary veins into the right atrium. There were one large and two small pulmonary valve cusps without fusion. The large valve cusp had been bisected by the valvulotome as had been a small cusp opposite it. There were linear scars in the infundibulum probably resulting from the passage of the valvulotome and the dilator. In spite of the complex malformation, catheterization and angiocardiographic studies demonstrated that the anomaly functioned essentially as a pulmonary stenosis with intact ventricular septum. It is of interest that marked improvement followed valvulotomy and dilatation and persisted during the thirteen months he lived.

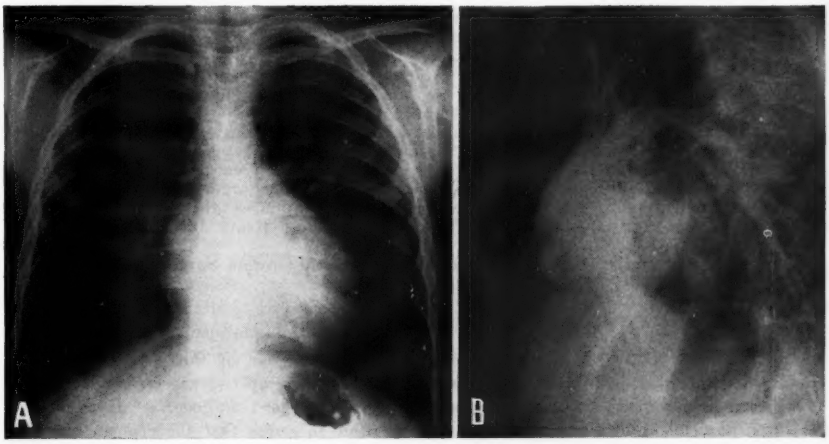

Fig. 5.—Roentgenograms in an unusual case of atypical tetralogy of Fallot. *A*, Plain roentgenogram showing right ventricular enlargement and diminished pulmonary vascular markings. *B*, Roentgenogram made shortly after intravenous injection of Diodrast. The pulmonary arteries are well visualized and there is no simultaneous opacification of the aorta.

SUMMARY

A new operative approach for pulmonary valvulotomy through a midline sternal splitting incision has been described. It gives excellent exposure of the anterior surface of the right ventricle and pulmonary artery and obviates the dislocation of the heart into the left thorax necessary when a left anterior thoracotomy is used in cases without marked right ventricular enlargement. A series of eight cases of pulmonic stenosis without evidence suggesting ventricular septal defect have been presented, and the diagnostic characteristics of this condition are presented with particular reference to the milder cases without the

classical signs noted in those with more advanced alterations. Good functional results were obtained even when the right ventricular pressure was not elevated to extreme levels and when mild pulmonary hypertension was present. The results of arterial oxygen and catheterization studies have been discussed. Three unusual cases of tetralogy of Fallot in which the patients were subjected to valvulotomy or pulmonary artery dilatation have also been presented.

Thus far there has been reported a total of sixty-two operatively treated patients with pulmonic valvular stenosis without ventricular septal defect, of which fifty-four survived and were benefited by the procedure.[2, 4-7, 9] Adding our eight cases in which no deaths occurred, seventy patients have been treated, with a mortality of 10 per cent. It is believed that the mortality will prove to be much lower in the milder cases of pulmonic stenosis and that it is worth while to continue exploration of the problem of their surgical treatment.

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DISCUSSION

DR. OSLER ABBOTT, Atlanta.—I am deeply appreciative of the physiologic understanding and thought involved in this paper. I want to bring up two aspects which I think are of some importance. One of the great features of a good surgeon, or of a surgical procedure, is adjustability, and I think it is important to stress that there is an optimum approach to every organ and structure. There is, however, a terrific adjustability in the thorax, and pulmonic stenosis can be approached from a lateral incision if the surgeon will adjust himself to moving from the left side of the table to the right, from an anterior incision and from a median sternotomy. Of course, much of the future of surgery is the proper combination of useful procedures, including the mixing of one incision with the other. I think that this is important. A tetralogy can be explored where it is planned to do, for instance in the young age group, a side-to-side anastomosis of the Potts type. The clinical judgment may not be found good, and a switch may be made to a valvulotomy, still using the lateral incision. With the lateral approach, if the surgeon will work facing the patient's anterior thorax as he lies in the left lateral decubitus, he will have good access.

The second point I would like to make is one on which I have had some correspondence with Dr. Blalock, and that is the question of anesthesia in pulmonary stenosis surgery. The question in my mind has been, if we give some positive pressure prior to opening the thorax, do we not increase pulmonary resistance, or alveolar tension, produce reflex pulmonary artery and bronchial artery spasm and therefore indirectly produce cardiac anoxia? In pulmonary

valve surgery the unfortunate frequency of cardiac standstill at the beginning of the operation is a phenomenon which so many of us have encountered, and Mr. Brock has so well emphasized. He insists that the surgical approach to these pulmonary stenosis patients must not be delayed and if difficulty develops, the valve must be opened quickly. The improvement of the patient is often striking as soon as the thorax is opened. Since that talk about three years ago, we have made a markedly strong point, that no positive pressure is given to a patient with obstruction of the right ventricular outflow tract until the thorax is opened. I would be interested to see what the results of such a concept would be if applied by a larger number of surgical clinics.

DR. LEO J. KENNEY, Detroit.—At the Henry Ford Hospital we have carried out a pulmonary valvulotomy in a total of ten cases, and there has not been any mortality in the series. We have a great interest in this subject.

I would like to present a simple diagnostic test devised by Dr. Robert Ziegler of the Division of Pediatric Cardiology, Henry Ford Hospital, which has given us considerable aid in the diagnosis of this entity and also in the diagnosis of the other congenital cardiac anomalies.

(Slide.) The test is carried out at the time of cardiac catheterization, after the physiologic data have been obtained. The tip of the catheter is permitted to remain in the right ventricle and 2 c.c. of 2 per cent sterile fluorescein dye is injected through the catheter into the right ventricular chamber. The period of time required for the dye to appear at the mouth is determined by a stop watch and an ultraviolet light directed at the mucous membrane of the patient's lips. A definite end point is obtained and identified by the characteristic appearance of fluorescein. Normally, four to five seconds are required for the dye to go from the right ventricle through the lungs, back into the left side of the heart, and out into the systemic circulation to the lips. In patients with pure valvular stenosis this time may be prolonged up to fourteen seconds. In the instance of a tetralogy of Fallot, with the patent interventricular septal defect, the dye appears in one to four seconds. This test is in routine use at our hospital and has been applied in over 200 cases without any harmful effects. Details have been published in a recent issue of the journal of *Circulation* by Dr. Ziegler.

DR. H. WILLIAM SCOTT, JR., Nashville.—I would like to congratulate Dr. Shumacker on these interesting studies and on his good results in these patients with valvular pulmonic stenosis. In the first direct attack on a stenotic lesion of a pulmonary valve, which was done at Johns Hopkins Hospital, Dr. Blalock used a sternum-splitting incision and the exposure was quite satisfactory. So far as I know, since that time exploration has been done in all subsequent cases through an anterior intercostal incision on the left. When the heart is large this exposure is quite satisfactory, but when it is small, as Dr. Shumacker has pointed out, there are some objections to this approach. It may be necessary, under these circumstances, to rotate the heart to the left by traction on the medial flap of the pericardium and that, certainly, is not entirely without ill effects. In such instances I have always had in mind the extension of the intercostal incision transversely across the sternum to gain adequate exposure of the base of the right ventricle and feel that with this additional maneuver, which may not be necessary except on rare occasions, the anterior intercostal approach is satisfactory for routine use, and in this use it may disturb the patient less than the use of a sternum-splitting procedure.

DR. HENRY SWAN, Denver.—I would like to put in an additional plug for the sternum-splitting maneuver. We have been splitting the sternum transversely in patients in whom it seemed desirable to have a wide exposure of the heart and access to all chambers. The incision has been in the fourth interspace on the right, entering the pleural cavity, across the sternum in a jog formation, and out the fifth interspace on the left. This little jog is put in to stabilize the postoperative repair of the sternum. We have encountered four patients with intracardiac foreign body, two with constrictive pericarditis, and one with an aneurysm of the ascending aorta through this incision, and it certainly provides a very wide and satis-

factory exposure. I would agree with Dr. Scott that in patients with pulmonic stenosis, if the left intercostal incision is placed, going across the sternum in cases in which it was found necessary, this would certainly give a very adequate exposure.

DR. RALPH A. DETERLING, JR., New York.—At Presbyterian Hospital in New York, in Dr. Humphreys's group, we have performed about twenty Brock operations with no mortality. We have employed the left anterior approach and have as yet had no great difficulty. I would agree with Dr. Swan and Dr. Scott in the desirability of being able to carry out a transsternal splitting if it did seem necessary. The main reason I wish to speak, though, is to mention the findings in two interesting cases we had in this group of pulmonic stenosis. At operation, direct intraventricular and arterial pressure are obtained by fine plastic catheters introduced through a small needle into the lumen and connected to a Lilly manometer. We have carried out perhaps 200 such studies in operations on the heart and great vessels, and have found several in which the findings at operation have been extremely important in influencing the surgical technique. In one of Dr. Humphreys's patients, the standard Brock procedure had been completed. A study of the pressures obtained with the catheter, about 1 mm. in diameter, demonstrated to our surprise that there was relatively little change in the pressure of the pulmonary artery, which was quite low, or in the elevated right ventricular pressure. So with some hesitation, as you can imagine, he entered the right ventricle and did a much more vigorous dilatation of the valve. The pressure tracings then indicated restoration toward normal of these pressures.

In a second patient upon whom I operated, we were studying the pressures at the end of the operation and were somewhat surprised to find the fall in the right ventricular pressure to be much less than was the rise in the pulmonary arterial pressure. Upon introducing the catheter into the right ventricle, I was informed by our associates that they had obtained an aortic pressure and pulse wave contour. The tip of the catheter had gone through an unsuspected high interventricular septal defect. Actually, the Brock procedure was allowing a more effective left to right shunt by opening the right ventricular outflow tract.

I think we should continue with such studies, since we are actually exploring the field of human cardiovascular physiology at the present time.

DR. SHUMACKER (Closing).—I certainly appreciate the comments of those who have discussed our paper. We have used the fluoroscein test which Dr. Kenny mentioned but have not found it entirely reliable. Certainly any procedure which would increase the accuracy of diagnosis would be of great value. One case, which is described in the manuscript but was omitted from the presentation for the sake of brevity, illustrates clearly the difficulty which one may encounter in diagnosis. This boy was eventually proved to have an atypical tetralogy of Fallot. According to cardiac catheterization studies and angiocardiography his malformed heart functioned in a manner quite like that of pulmonic stenosis and he was considerably improved after pulmonary valvulotomy and dilatation of the infundibulum and the pulmonary artery.

I am sure that the comments which have been made about the extensibility of various thoracic incisions are sound. We too have done explorations on several patients through lateral thoracotomies and, when we found it necessary to open the pericardium and perform pulmonary valvulotomy, have simply extended the incision anteriorly and completed the procedure satisfactorily. We are at present using the sternum-splitting approach in seemingly proved cases of pulmonic valvular stenosis because we find that it is so easy in execution, that it provides such excellent exposure, and is so easy to repair. It seems to me that it is an approach which others may profitably employ in cases of this sort. It is of great interest to me to learn that Dr. Blalock used it in his first case of pulmonary valvulotomy.

PLASMACYTOMA OF THE LUNG

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CHARLOTTESVILLE, VA.

EXTRAMEDULLARY plasmacytoma is a relatively rare tumor and in the literature there exists much confusion concerning its origin and behavior. A survey of the literature shows that about 185 cases of extramedullary plasmacytoma have been reported since 1905. Hellwig[1] reviewed 127 cases in 1943 and added one of his own. This represented all cases reported from 1905 to 1943, with the exception of 24 cases reported by Jaeger[2] in 1942. Since that time a number of cases have been added and several reviews of the subject have been presented.

In all of these, only four extramedullary plasmacytomas were reported which were thought to have originated in the lung. The first was reported by Gordon and Walker in 1944.[3] There is little doubt that this is a plasmacytoma of the lung. The patient survived left upper lobectomy, but there is no further report. The second patient with a plasma cell tumor believed to be in the lung was reported by Kuley and Kuntman.[4] The lesion in their patient, however, is not conclusively in the lung. The roentgenograms presented with the report show that the lesion could well have originated from the mediastinum. The diagnosis rests only upon material obtained by aspiration; therefore, it is difficult to accept this as a plasmacytoma of the lung. The third report by Childress and Adie[5] describes a plasmacytoma in the right middle lobe of a 21-year-old white man. It was removed by segmental resection. At the time of their report the patient had been followed for three years and apparently was doing well. The fourth report by Cotton and Penido[6] describes a 57-year-old woman who survived left upper lobectomy for plasmacytoma and who was well some twenty months later at the time of the report.

Because of the obvious rarity of this condition, and because of other unique features, it was thought useful to report a plasmacytoma of the lung which came under our observation. We believe this to be the fourth confirmed plasmacytoma of the lung. It is the youngest patient (age 3 years) on record with extramedullary plasmacytoma in any location. It represents the longest follow-up (eight years) of any plasmacytoma of the lung.

CASE REPORT

History.—D. C. S., University of Virginia Hospital No. 202614. This 3-year-old white girl was admitted to the Pediatric Service at the University of Virginia Hospital on June 6, 1944, with generalized swelling and dyspnea of one year's duration. She had been well for the first two years of her life, and one year prior to entering the hospital she became fretful and lost her appetite. Later she developed generalized edema, and her urine became dark. These signs persisted until admission. During the year she had several bouts of fever accompanied by increase in the edema. She also had exertional dyspnea which became progressively worse. During the winter months, the patient had developed an intolerance

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Received for publication, June 6, 1952.

to cold, and became cyanotic on the slightest exposure to cold. Six months prior to admission, the parents noted clubbing of the fingers which became more prominent until admission. There was no cough, hemoptysis, expectoration, or specific chest pain. Her past history revealed that she had been born by a normal delivery, had never been cyanotic as an infant, had an entirely normal development, and was very energetic until the onset of her illness.

Physical examination on admission revealed a temperature of 103.6° F. rectally, pulse rate of 120, respirations 40. She weighed 14.6 kg., and appeared to be a well-developed 3-year-old girl with 2 plus pitting edema of the arms, legs, and face. She was in no distress. There were shotty, nontender, discrete nodes in all of the regional areas. The spine showed a moderate dorsal kyphosis. There was a moderate increase in the anteroposterior diameter of the chest, and respirations were almost entirely abdominal. There was dullness to percussion over the left hemithorax from the third rib anteriorly to the third thoracic vertebra posteriorly, with decreased breath sounds over the same area. There were no râles. Examination of the heart revealed a diffuse forceful impulse, most intense below the sternum at the level of the fifth costal cartilage, which gave the impression that the heart was pushed to the right. There was a harsh systolic murmur over the entire precordium. The neck veins were distended, and there were many enlarged cutaneous veins over the thorax, arms, and legs. The liver edge was 2 to 3 fingerbreadths below the right costal margin. The spleen was readily palpable. There was slight abdominal distention. The knees were swollen, fluctuant, and painful on motion. There was marked clubbing of the fingers and toes.

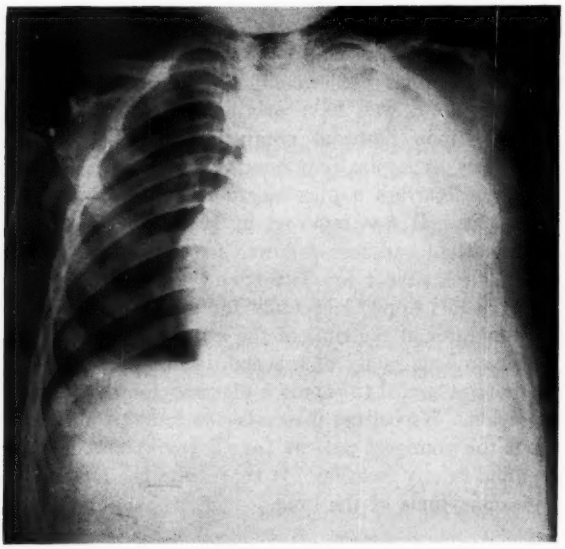

Fig. 1.—Preoperative roentgenogram showing anteroposterior view of dense mass occupying nearly the entire left hemithorax.

Laboratory Data.—Hemoglobin—8 Gm. (Haden-Hauser); red blood cells—4.3 million; white blood cells—7,600, with a normal differential. The venous pressure in the right arm was 150 mm. of water. The blood Wassermann and Kahn were negative. Blood urea was 22 mg. per 100 c.c. Total protein was 5.9 Gm. per cent, and a single blood culture was negative at forty-eight hours. The urine was yellow, slightly cloudy, with 4 to 8 white blood cells and 10 to 15 red blood cells per high-power field, with no casts. It was negative for Bence-Jones protein.

Roentgenograms of the chest revealed a very large rounded opacity which nearly filled the left hemithorax. The mass occupied the posterior portion of the chest to within 1 to 1.5 cm. of the anterior thoracic wall (Fig. 1). There were no definite pulsations on fluoroscopy. The

heart and most of the mediastinum were quite markedly displaced to the right. Roentgenograms of the extremities revealed almost the total length of the shafts of all the long bones to present marked periosteal new bone formation which was sharply demarcated from the underlying cortex. The peripheral margins of the bones were also sharply delineated. The mass suggested the possibility of a large fluid-filled pulmonary cyst, a walled-off pocket of fluid, or possibly a huge intrathoracic neoplasm. The bone changes were typical of a marked degree of pulmonary osteoarthropathy.

Aspiration biopsy of the mass was performed. Examination of the material obtained revealed a solid tumor made up of large masses of spindle-shaped cells with hyperchromatic nuclei. Mitoses were thought to be moderately increased. Some palisading of the cells was noted. The exact type of tumor could not be determined from such a minute biopsy, but it was thought that the tumor might represent a sarcoma or lymphoblastoma.

It was thought from the histologic appearance that the tumor might respond to x-ray treatment. This treatment was delayed because the patient developed measles, which subsided in four days. Three days later roentgen therapy was begun over the left hemithorax; 2,800 r in air (filtration—2 mm. Cu) being given through two portals over a period of fourteen days. Following this a roentgenogram of the chest revealed the tumor to have increased in size and to have extended to the first interspace. Because of this failure to respond to therapy, it became obvious that thoracotomy offered the patient her only chance for cure. On July 10, 1944, exploratory thoracotomy was carried out by one of us (M. L. W.). The pleura was freed, and a large dense mass was found occupying all of the lower lobe on the left and extending across the interlobar fissure into the upper lobe. There appeared to be no normal lower-lobe tissue remaining. Exploration of the mediastinum revealed it to be entirely free of tumor. Pneumonectomy was carried out using the individual ligation technique. One lymph node was removed for examination, and grossly appeared benign. It was felt that the entire tumor mass had been removed. The patient tolerated the procedure nicely, and had an essentially uneventful postoperative course.

On gross examination, the tumor in the left lower lobe measured 7 by 7 by 8 cm. It was yellowish-brown and fairly soft. It seemed to have a definite capsule, and was quite circumscribed on cut section. It did not appear to arise from a bronchus. The hilar region was not involved. On microscopic examination, the tumor was composed of a stroma of spindle-shaped cells growing in loose strands. These cells had blunt, rounded nuclei, and were fairly uniform in structure and staining properties. The predominant cells, however, in most fields were round, oval, or polygonal cells with abundant cytoplasm and eccentric nuclei containing from five to ten distinct deeply-staining blocks of chromatin regularly arranged in a circle within the nuclear membrane. Adjacent to the nucleus there could be found the characteristic "paranuclear light-stained" area. This predominating cell fulfilled the criteria for the diagnosis of plasma cells as outlined by Marschalko.[7] A search was made for Russell bodies but none were found (Fig. 2).

The patient's postoperative course was essentially uneventful. She was discharged on Aug. 6, 1944, in good condition. She first returned for follow-up examination on Oct. 2, 1944. There was an opacity in the left hemithorax, with a fluid level and thickening of the pleura. The periosteal new bone formation and the obvious signs of advanced osteoarthropathy were noted to be subsiding. The patient was then asymptomatic, and gained weight for one year. A small mass in the right lung was noted in 1945 and was followed. By Jan. 31, 1946, it was noted to have decreased in size and become less distinct. It was thought to be a typical Ghon tubercle. The osteoarthropathy had completely disappeared by Jan. 1, 1946. Radiographic follow-up of the chest during and subsequent to 1946 show clearly that there were lung markings filling the entire chest and on examination the patient had good breath sounds over the operative side (Fig. 3). At last examination (March, 1952) the patient was entirely well. She was able to run and play with other children and had no detectable respiratory limitations. Her respiratory studies show a maximum breathing capacity of 30.8 L per minute, with a vital capacity of 1,346 c.c. The patient is gaining weight and doing well in school. Expansion of the operated side on respiration is good, and nearly equals that of the right side. The breath sounds on both sides are equal. Filling of the entire thorax by the remaining lung following pneumonectomy has been noted in other patients. It is unusual, however, to find so little distortion in the mediastinum.

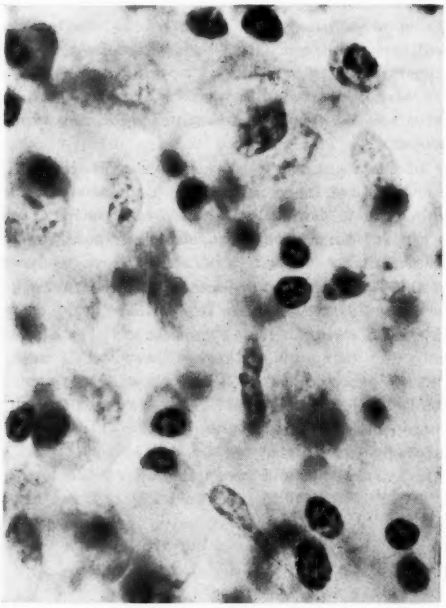

Fig. 2.—Photomicrograph (hematoxylin-eosin ×1200) showing the predominating typical Marschalko plasmacytes.

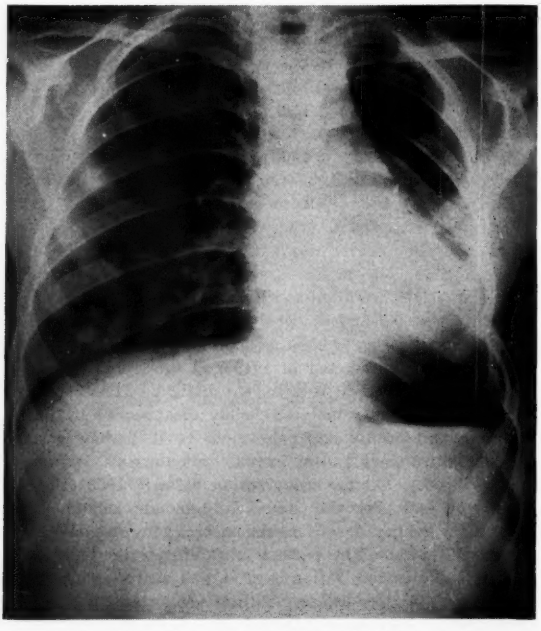

Fig. 3.—Roentgenogram showing anteroposterior view of thorax eight years after left pneumonectomy. Faint but definite lung markings can be seen in the left hemithorax.

DISCUSSION

Recent work dealing with the plasma cell by Jordan[8] has established some new information concerning the nature of the plasma cell, and has confirmed previous data dealing with the origin of this cell. Studies by Jordan of tissue explants, consisting almost solely of lymphocytes, have clearly shown that lymphocytes differentiate into plasmacytes. In many tissue explants containing lymphocytes, the predominating cells after five to six days were typical Marschalko plasmacytes. In addition, the plasmacytes were found capable of extensive ameboid motility and complete amitotic division. The latter was confirmed in cinematographic records. These actual cinematographic records confirm the tissue culture work done by Maximow[9] in 1922, which showed the derivation of plasma cells from lymphocytes. In spite of Maximow's work, there has been considerable argument concerning the origin of the plasma cell. Originally, Ramon y Cajal, who first described the plasma cell in 1890, contended that the plasma cell originated from fixed tissue lymphocytes. Unna contended that plasma cells are derived from fixed connective tissue elements. Marschalko believed the origin of plasma cells to be from emigrated blood lymphocytes only. Maximow and Jordan's works appear to present definite evidence that plasma cells may be derived from both fixed and emigrated lymphocytes. Since the plasmacytoma may originate from lymphocytes, it may bear some relation to tumors of lymphatic tissue.

It has been maintained by some that extramedullary plasmacytomas are granulomatous in nature, and consequently are benign. However, a review of the clinical experience with these tumors shows this to be a dangerous viewpoint. Hellwig found that out of 128 cases reported in his review, 29 showed definite invasion locally. Thirteen of these showed distant metastases. Figi, Broders, and Havens[10] reported eleven cases of plasmacytoma in 1945. Five of the eleven cases showed frankly malignant behavior with either extensive local invasion or distant metastases. On the basis of their experience they have stated: "Plasmacytomas are malignant lesions and they should be classified and treated as such." They found no evidence from their material to suggest that these lesions are granulomatous or inflammatory in nature. In 1946, Couret[11] reported a plasmacytoma of the pyloric portion of the stomach. This lesion was locally resected. It was found to be a plasmacytoma, and was thought to be an inflammatory benign lesion. Two months later the patient was explored for obstruction, and was found to have large fungating masses throughout the abdomen which proved to be composed predominantly of lymphocytes. One month later, autopsy revealed the large definitely invasive masses to represent plasmacytoma. Shaw and Smith[12] reported a plasmacytoma of the thyroid gland which was almost exclusively made up of plasma cells with a moderate number of mitoses suggesting a low-grade malignancy rather than an inflammatory lesion. Many other cases can be cited which show definitely that a high percentage of plasmacytomas become malignant locally and give rise to distant metastases. It should be stressed that the microscopic picture of these tumors cannot be correlated with their clinical behavior. These cells have been shown to be capable of amitotic division and in tissue cultures have been noted to multiply by this means in

most instances rather than by mitotic division. It is easy to see, therefore, why the absence of mitoses in these tumors has misled many observers into classifying the lesion as benign, only to have the lesion become frankly malignant. It is obvious that plasmacytomas cannot be classified on the basis of their microscopic appearances in the same manner as carcinomas.

Gross appearance may also be misleading. Apparent encapsulation and sharp delineation from surrounding tissue have been noted in lesions which have given rise to metastases. Because plasma cells have been noted to be abundant in some inflammatory diseases, particularly syphilis, tuberculosis, and neisserian infections, many observers have regarded practically all lesions containing plasma cells as granulomas. These inflammatory lesions should be sharply separated from plasmacytomas such as the one reported here, in which there is little or nothing to suggest an inflammatory lesion.

TREATMENT

Sixty-five per cent of the reported cases have been treated by surgery alone, about 18 per cent have been treated by a combination of surgery and radiation, and the remainder have been treated by radiation alone. Some of the tumors reported were apparently radiosensitive and good results were obtained but a number of others failed to respond to roentgenotherapy as did our case. Surgical treatment was then utilized when it was still feasible. In view of the good results obtained in our one patient and the good immediate result obtained by surgical means in the other three plasmacytomas of the lung, surgery would certainly appear to be the treatment of choice when the lesion is pulmonary in origin. According to the results obtained in treated cases reported in the literature, it would seem that surgery is also the treatment of choice in those tumors elsewhere in the body that are accessible to removal. A number of these tumors have recurred, however, despite surgery and radiation combined. Apparently the best results are obtained in tumors located in the conjunctiva. The conjunctival tumors are benign lesions with few exceptions, though Tajkef[13] reported a case of plasmacytoma of the conjunctiva which recurred repeatedly after several excisions. It has probably been these tumors of the conjunctiva that have led some workers into a misconception concerning plasmacytomas. In other locations in the body, the tumors have by no means been benign. The experiences of Couret, Shaw and Smith, Figi and Broders, and others testify to the highly malignant behavior of a large percentage of extramedullary plasmacytomas. Certainly, it is safe to say that our knowledge of these tumors is limited, and at present there is no means of determining in an individual case whether a plasmacytoma is benign or malignant provided, of course, metastases are not already obvious. Until we are able to make this determination in a given case, we are inclined to agree with Figi and Broders that these tumors should be regarded as malignant and treated accordingly.

SUMMARY

A case of plasmacytoma of the lung in a 3-year-old girl is presented. A left total pneumonectomy was done, and the patient has been followed for eight

years and has no evidence of recurrence or metastasis. This is apparently the fourth reported case of plasmacytoma of the lung proved clinically and pathologically. A follow-up of eight years is presented with complete regression of advanced pulmonary osteoarthropathy and complete filling of the thorax by the remaining right lung. A brief review of the literature is presented in which it is pointed out that these tumors in many instances are malignant. Surgical removal is the treatment of choice in most cases of extramedullary plasmacytomas outside the conjunctiva, provided there has been no metastasis or invasion of vital structures.

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PHYSIOLOGIC STUDIES FOLLOWING THORACIC SURGERY

II. Immediate Effects of Upper Lobectomy Combined With a Five-Rib Thoracoplasty

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INTRODUCTION

THE first paper in this series reported on the immediate physiologic effects following each of three stages of a seven-rib thoracoplasty.[1] The present work deals with a similar study made following a one-stage operation in which an upper lobe was resected and a five-rib thoracoplasty was performed.

In the search for a satisfactory procedure for the treatment of cavitary and nodular active tuberculosis, many surgical approaches are being tried by various groups. The combination of a five-rib thoracoplasty and an upper lobectomy was devised to allow extirpation of the principal site of the disease without overexpansion of the lung tissue remaining in the chest.[2-4] The thoracoplasty was performed immediately after the lobectomy and as a part of the same procedure. A subsequent report will cover the effects of segmental and wedge resections. Since resectional procedures are relatively new, it was felt advisable to obtain accurate observations upon patients subjected to such operations and to compare the effects of the various procedures with the effects of a thoracoplasty. Thoracoplasty was selected as a standard for comparison, since the clinical effects of this operation are so well known.

It is to be emphasized that this study was concerned only with the immediate physiologic effects of the various procedures. No consideration was given to the effects upon the tuberculous disease.

METHODS

The methods of study employed in this group of patients were the same as those reported in detail in the first paper of this series.

CLINICAL MATERIAL

Eleven male patients ranging in age from 20 to 59 years were selected for study. Each had previously been reviewed and accepted for surgery by members of the medical and thoracic surgical services. Each patient had predominantly unilateral, upper lobe, cavitary and nodular tuberculosis. The contralateral lung was either clear or contained apparently stable tuberculous lesions. In one case (N. H.) there was pleural thickening over the lower and middle lobes on the involved side as a result of a previous therapeutic pneumothorax.

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Published with approval of Chief Medical Director. The statements and conclusions published by the author are the result of his own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

Received for publication, June 10, 1952.

In this patient the middle and lower lobes were decorticated at the time of the lobectomy-thoracoplasty. In one subject (H. R.) the right middle lobe was also removed because of nodular disease. In this same case there was evidence of pleural thickening over the contralateral lung as a result of a previous therapeutic pneumothorax. This case will be discussed in detail below. One patient (L. D.) had silicosis (Stage II) throughout all five lobes, but functionally he did not constitute a special problem. Only one patient (W. H.) manifested any dyspnea on the exertion of ascending a flight of stairs. The comparison of the preoperative pulmonary functional studies with those of a group of 16 normal residents is given in Table I. The only significant abnormalities were a decrease in maximum breathing capacity, an increase in alveolar retention of nitrogen after seven minutes of breathing pure oxygen, and an increase in the residual volume (Table I).

TABLE I. THORACOPLASTY AND LOBECTOMY

	MEAN NORMAL	MEAN PREOPERATIVE	MEAN POSTOPERATIVE
Oral temperature (°F.)		98.2 ± 0.55	100.3 ± 1.25*
Pulse rate/min.		78.0 ± 12.5	116 ± 16.8*
Metabolic rate $\frac{\text{(cal./hr.)}}{M^2}$	35.0 ± 2.7	38.8 ± 5.0	47.0 ± 5.7*
Respiratory quotient	0.90 ± 0.043	0.80 ± 0.08	0.76 ± 0.05
Arterial blood:			
pH	7.40 ± 0.01	7.39 ± 0.01	7.37 ± 0.01
CO_2 content mm./L.	21.30 ± 1.0	22.37 ± 0.95	22.49 ± 2.4
P_{CO_2} (mm. Hg)	39.0 ± 2.0	43.3 ± 3.0	46.0 ± 6.1
Hemoglobin saturation (per cent)	96.5 ± 0.82	96.0 ± 1.09	88.3 ± 6.5*
Pulmonary shunt (per cent)	0	0.5 ± 1.0%	15.0 ± 1.1%*
V (c.c./min.)	5641 ± 601	5843 ± 860	8405 ± 2850*
f (Respirations/min.)	12 ± 1.82	13 ± 3.6	22 ± 9.4*
V_T (c.c.)	499 ± 58	470 ± 105	384 ± 61.1*
V_D (c.c.)	144 ± 32	152.7 ± 30.2	161.5 ± 51.0
$\dot{V}_D$ (c.c./min.)	1605 ± 421	1712 ± 543	3743 ± 2200*
V_A (c.c.)	310 ± 47.4	271.4 ± 87.9	176.0 ± 46.0*
$\dot{V}_{CO_2}$ (c.c./min.)	212.3 ± 23	203 ± 20.9	237 ± 36.3*
$\dot{V}_A$ (c.c./min.)	3496 ± 381	3261 ± 459	3695 ± 1149*
Vital capacity c.c./M^2	2290 ± 274	2210 ± 290	
Residual volume c.c./M^2	680 ± 148	934 ± 165†	
Reserve volume c.c./M^2	462 ± 116	510 ± 138	
Total lung volume c.c./M^2	2970 ± 313	3150 ± 283	
RV/TLV per cent	23.0 ± 4.1	29.9 ± 5.1†	
Index pulmonary mixing (per cent N_2)	1.45 ± 0.23	2.53 ± 0.85†	
Maximum breathing capacity L./min./M^2	60.7 ± 5.7	48.8 ± 7.3†	

*Statistical analysis reveals this mean to differ significantly from the preoperative mean ($p = 0.05$ or less).

†Statistical analysis reveals this mean to differ significantly from the mean value obtained in 16 normal subjects ($p = 0.05$ or less).

The resting minute volume of ventilation and basal metabolic rates did not differ significantly from the normal values. The arterial blood hemoglobin saturation, carbon dioxide content, carbon dioxide tension, and pH were also

within normal limits. The mean for the oxygen dissolved in the arterial blood by breathing 100 per cent oxygen was not significantly different from the mean value found in normal subjects. From these data the portion of blood being shunted through nonaerated pulmonary tissue was found to be normal preoperatively. This is consistent with the findings reported recently by Ebert.[5]

RESULTS

The mean results of the pre- and postoperative studies are recorded in Table I, and the changes illustrated in Figs. 1 and 2.

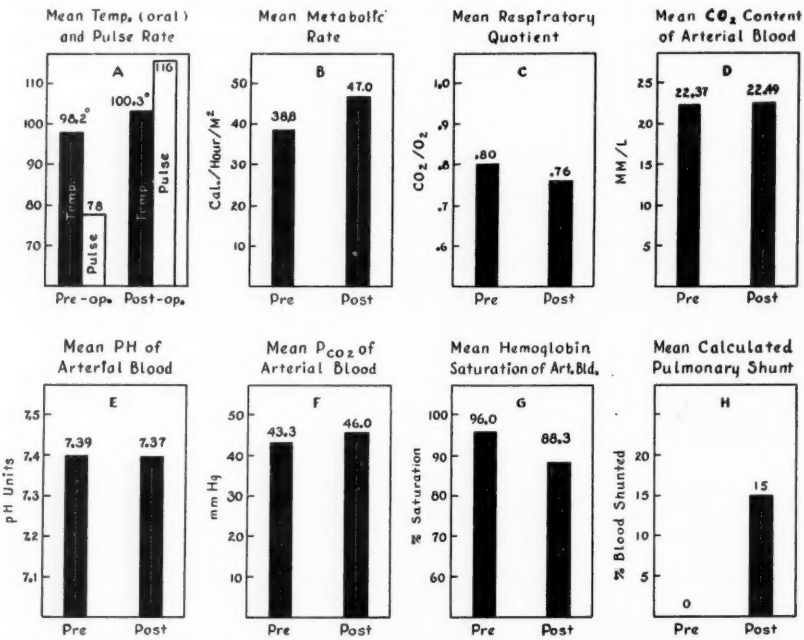

Fig. 1.—Physiologic changes following lobectomy-thoracoplasty (sixteen to twenty hours after surgery). Comparison of the preoperative and postoperative means.

The mean oral morning temperature rose from 98.2° to 100.3° F. the day after surgery (Fig. 1, *A*). The mean preoperative pulse rate was 78 per minute, as compared with a mean of 116 per minute postoperatively (Fig. 1, *A*). The mean postoperative metabolic rate was 38.8 cal./hr./M² preoperatively and 47.0 cal./hr./M² postoperatively (Fig. 1, *B*). The above three changes were all statistically significant. The respiratory quotient remained essentially unchanged pre- and postoperatively (Fig. 1, *C*).

The mean carbon dioxide content of arterial blood was 22.37 mm./L. preoperatively and 22.49 mm./L. postoperatively (Fig. 1, *D*). The pH of the arterial blood was 7.39 before surgery and 7.37 on the morning following the operation (Fig. 1, *E*). The mean P_{CO_2} was 43 mm. Hg preoperatively and 45 mm. Hg postoperatively. None of these changes proved to be of significance.

The mean arterial hemoglobin saturation while the patient was breathing ambient air fell from 96.0 per cent preoperatively to 88.3 per cent postoperatively (Fig. 1, *G*). This change was of statistical significance.

The quantity of oxygen physically dissolved in the arterial blood by exposure to an alveolar oxygen tension of about 600 mm. Hg by the breathing of 100 per cent oxygen fell from 1.99 volume per cent preoperatively to 1.22 volume per cent postoperatively. From this fact one can calculate that a 0.77 volume per cent fall occurred in the oxygen content of the blood leaving the lungs. Such a fall was probably a result of admixture of blood passing through nonaerated lung tissue. This change represents a 15 per cent functional pulmonary shunt (Fig. 1, *H*). This degree of postoperative pulmonary shunt would account for about one-half of the unsaturation of arterial blood which was noted when the patient breathed ambient air.

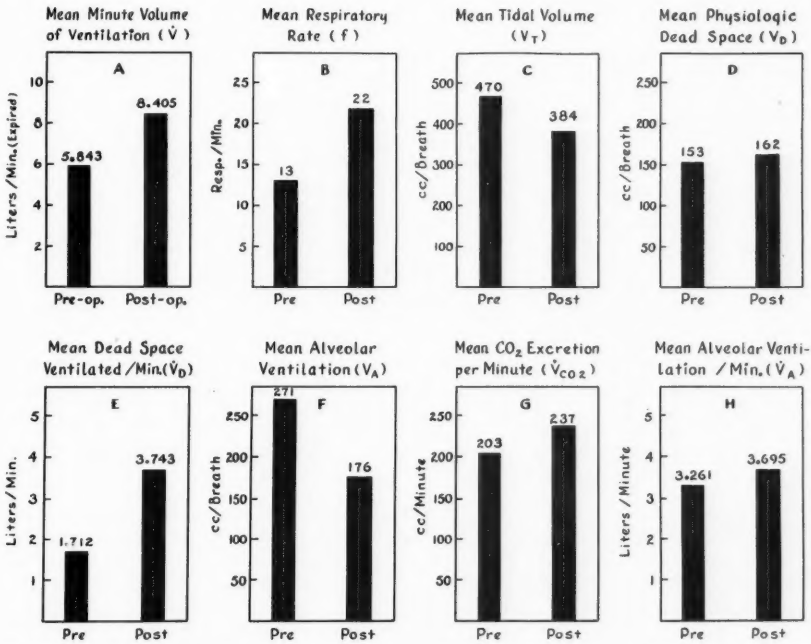

Fig. 2.—Physiologic changes following lobectomy-thoracoplasty (sixteen to twenty hours after surgery). Comparison of the preoperative and postoperative means.

The minute volume of ventilation rose from a preoperative mean of 5,843 c.c./min. to 8,405 c.c./min. on the morning following surgery (Fig. 2, *A*). This change was accounted for largely by an increased rate of respiration from 13.0 to 22.0 per minute (Fig. 2, *B*). The tidal volume fell from a mean of 470 c.c. preoperatively to a mean of 384 c.c. postoperatively (Fig. 2, *C*). These three changes were all highly significant. The volume of physiologic dead space rose significantly (153 c.c. preoperatively and 162 c.c. postoperatively) (Fig. 2, *D*).

The volume of dead space ventilated each minute (physiologic dead space times the respiratory rate) rose from a mean of 1,712 c.c./min. preoperatively to 3,743 c.c./min. postoperatively, a mean rise of 92 per cent (Fig. 2, *E*). This change was highly significant. Alveolar ventilation per breath decreased significantly from 271 c.c. before surgery to 176 c.c. shortly thereafter (Fig. 2, *F*). The rate of carbon dioxide excretion by the lungs rose from a mean of 203 c.c./min. preoperatively to 237 c.c./min. postoperatively (Fig. 2, *G*). From this the calculated alveolar ventilation per minute was found to be increased significantly from a mean of 3,261 c.c./min. preoperatively to 3,695 c.c./min. on the morning after surgery (Fig. 2, *H*). Both of these alterations were statistically significant.

Comparison of These Effects With Those of Thoracoplasty.—The mean physiologic effects of upper lobectomy in combination with a five-rib thoracoplasty were compared statistically with the mean effects rendered by each stage of a seven-rib thoracoplasty (reported in the first paper of this series). Thoracoplasty was used for comparison because its clinical effects are so well known that it can serve as a standard by which the effects of other surgical procedures can be judged. A lapse of sixteen to twenty hours was allowed after surgery before making the postoperative observations in order to allow for the compensation of the effects of general anesthesia (pentothal-curare) used in the

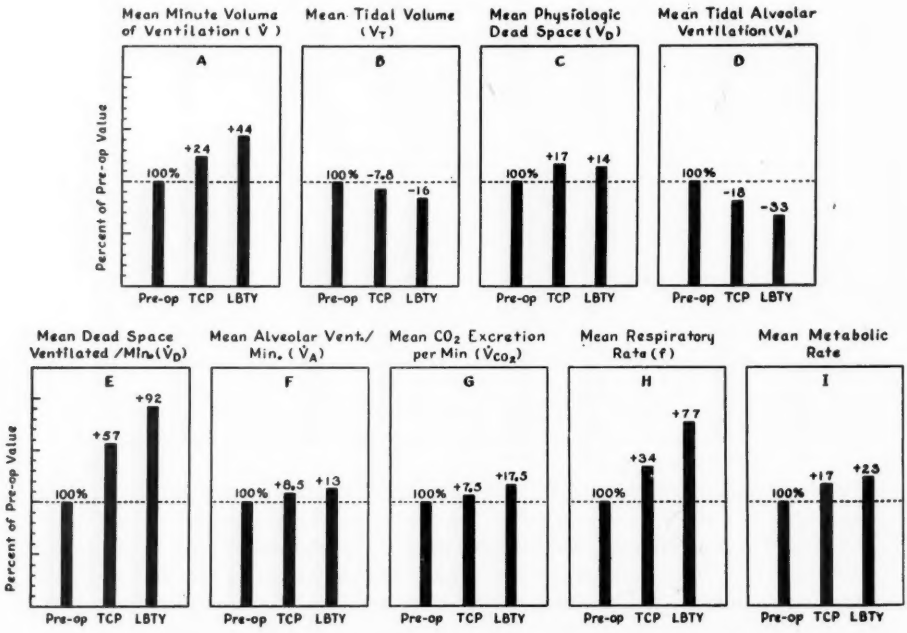

Fig. 3.—Comparison of the physiologic changes following lobectomy-thoracoplasty with those following thoracoplasty alone. The first bar represents 100 per cent of the preoperative value for each patient individually. The second bar represents the weighted mean percentage change of the three postoperative determinations in ten patients undergoing a three-stage, seven-rib thoracoplasty.

present series of patients, since only local anesthesia was used in the thoracoplasty series. The data collected after such compensation should be comparable to those obtained after thoracoplasty. The means of the percentage change from the individual preoperative values were compared for the two groups by the use of the t test. The most significant data are presented in Fig. 3 in which the postoperative changes following the two procedures are compared with the mean preoperative values as 100 per cent.

The increase of 44 per cent in the respiratory minute volume following lobectomy-thoracoplasty was significantly greater than the increase of 24 per cent following each stage of thoracoplasty alone (Fig. 3, *A*). Other measurements which showed a statistically greater alteration in the present series over those found in thoracoplasty were: (1) a fall of 16 per cent in tidal volume (Fig. 3, *B*); (2) a fall of 33 per cent in tidal alveolar ventilation (Fig. 3, *D*); (3) a rise of 92 per cent in volume of dead space ventilated per minute (Fig. 3, *E*); (4) a rise of 13 per cent in minute alveolar ventilation (Fig. 3, *F*) associated with an increase of 17.5 per cent in the elimination of carbon dioxide (Fig. 3, *G*); (5) a rise of 77 per cent in respiratory rate (Fig. 3, *H*); and, (6) a rise of 28 per cent in metabolic rate (Fig. 3, *I*). There was a small rise in physiologic dead space (Fig. 3, *C*) (14 per cent) which was similar to the change observed after each stage of thoracoplasty. This is probably attributable to the increased rate of respiration.

The rise in the temperature and pulse rate was significantly greater in this series of patients than in the patients undergoing thoracoplasty. Carbon dioxide content, pH, and carbon dioxide tension of the arterial blood were not altered significantly in either series sixteen to twenty hours after completion of the surgery. This fact gives evidence that the effects of general anesthesia had been compensated since it has been shown that respiratory acidosis due to CO_2 retention occurs during thoracic surgery performed under general anesthesia.

These data indicate that the lobectomy-thoracoplasty combination was of significantly greater magnitude than one stage of a thoracoplasty.

CASE REPORT

One death occurred in the present series, and it seems worth while to consider the data obtained in detail. The patient, H. R., was a white man aged 41 years. *History:* Bilateral upper lobe infiltrate was first discovered on separation roentgenogram in 1944. Sputum was found to be positive for tubercle bacilli. He was asymptomatic except for mild ease of fatigue. He was hospitalized and treated with bed rest from June, 1944, to June, 1946, when an artificial pneumothorax was induced on the left. There were no complications with the induction, but pleural fluid began to accumulate after two years. The sputum became negative thereafter, and he was discharged from the hospital in November, 1947. Pneumothorax was continued on an outpatient basis, and he returned to school. In October, 1949, there was a recurrence of ease of fatigue accompanied by pain in the right side of the chest. He was admitted to this hospital at that time. Several gastric washings had been positive since 1947. *Physical Examination:* He was well developed and well nourished, and was in no distress. Representative roentgenograms are shown in Fig. 4. At the time of surgery there was evidence of a bilateral nodular infiltrate and some pleural thickening on the left. No pneumothorax remained on the left. Planigrams of the right lung revealed multinodular infiltration with cavitation. The left side showed pleural thickening. The vital capacity

in February, 1951, was 4,200 c.c. and was rapidly expelled. Differential bronchospirometry revealed that the left lung accounted for 35 per cent of the total ventilation and oxygen absorption. The patient was treated with bed rest from October, 1949, to March, 1951. No chemotherapy was used during this period.

On March 19, 1951, resection of the right upper lobe was attempted. Because of extensive tuberculous involvement of the middle lobe, it was also sacrificed, and a five-rib posterior thoracoplasty was performed to close the dead space. The patient was dyspneic from the time of the surgery, and the respiratory rate ranged from 40 to 48 per minute. Oxygen therapy gave little or no relief of the dyspnea, but did relieve the cyanosis. On March 22, 1951, there was a sudden onset of auricular fibrillation, but the normal sinus rhythm was restored by the intravenous administration of Cedalanid. He showed occasional episodes of disorientation. He became moderately cyanotic when breathing room air. Drainage catheters were removed on the fourth postoperative day, and the wound was noted to be healing

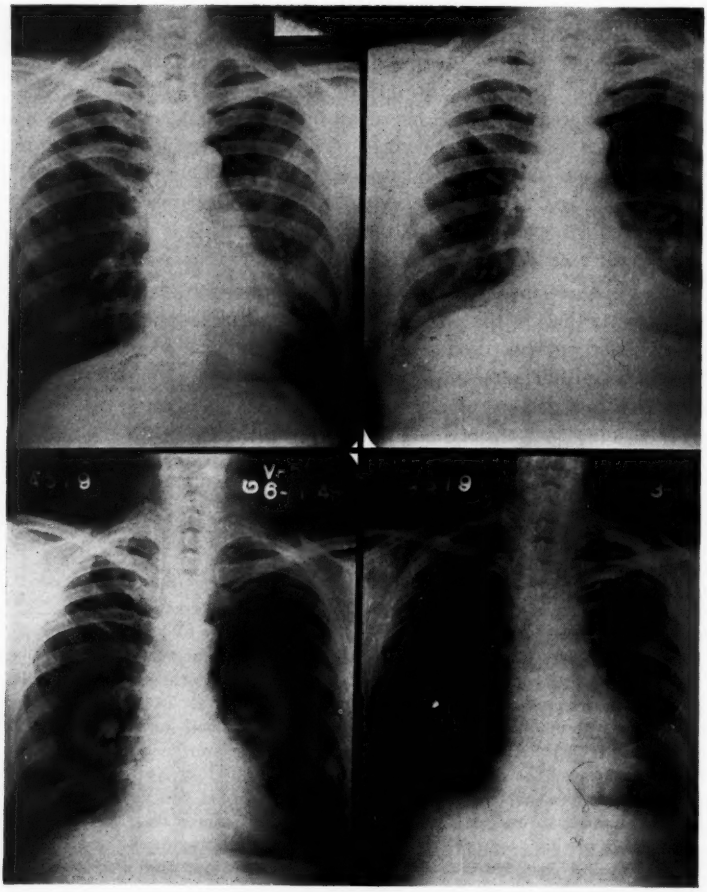

Fig. 4.—Case H. R. *Upper left,* film taken in November, 1944; *upper right,* film taken in March, 1947; *lower left,* June, 1949; and *lower right* shortly before surgery in March, 1951. The left pneumothorax is seen in the 1949 film, and the pleural residuals are brought out in the film of March, 1951. The nodular disease in the right upper and middle lobes was better demonstrated by planigraphy.

normally. Dyspnea continued to be so severe that simple conversation caused fatigue. On the evening of March 28, 1951, he received visitors for ninety minutes and seemed cheerful, in spite of the dyspnea. As the visitors left he commented to the nurse that he felt "all tired out." Ten minutes later he was found unconscious with very weak gasping respirations and a dusky gray cyanosis. He expired within a few minutes. The pulse was noted to be present after he had lost consciousness, but there was no mention of its character.

TABLE II. CASE H. R.—44 W. M.

	PREOPERATIVE	POSTOPERATIVE	PER CENT CHANGE
Oral temperature (°F.)	97.80	101.20	+3.5
Pulse rate per minute	82.00	126.00	*+53.7*
Metabolism rate (Cal./hr./M²)	34.70	48.60	*+40.1*
Respiratory quotient	0.80	0.76	−5.0
Arterial blood: pH	7.39	7.34	−0.7
CO_2 content mm./L.	24.28	20.79	−14.4
P_{CO_2} (mm. Hg)	47.00	45.00	−4.2
Hemoglobin saturation (per cent)	94.20	84.9	−9.9
Pulmonary shunt (per cent)	0.00	11.90	
$\dot{V}$ (c.c./min.)	6,045.00	14,270.00	*+135.4*
f (resp./min.)	15.00	46.00	*+206.0*
V_T (c.c.)	403.00	311.00	*−22.8*
V_D (c.c.)	150.20	171.20	*+14.0*
$\dot{V}_D$ (c.c./min.)	2,253.00	7,875.00	*+249.5*
V_A (c.c.)	206.80	93.80	*−113.0*
$\dot{V}_{O_2}$ (c.c./min.)	20.90	281.00	*+34.4*
$\dot{V}_A$ (c.c./min.)	3,101.00	4,313.00	*+39.1*
Vital capacity c.c./M²	1,759.00		
Residual volume c.c./M²	963.00		
Reserve volume c.c./M²	280.00		
Total lung volume c.c./M²	2,722.00		
RV/TLV per cent	35.40		
Index pulmonary mixing (per cent N_2)	4.25%		
Maximum breathing capacity L./min./M²	40.62		
Surface area (M²)	2.16		

Physiologic measurements were obtained before surgery and sixteen hours after the completion of the eight-hour surgical procedure. These data are given in Table II. It can be seen that the arterial blood showed no dangerous abnormality at the time of the postoperative study (sixteen hours after surgery). The amount of carbon dioxide excreted was increased in association with a slight fever. This stimulated an increased alveolar ventilation. The tidal volume was limited to 311 c.c. instead of the preoperative value of 403 c.c. In association with these alterations there was a rise in the respiratory rate from 15 to 46 per minute, which resulted in maintenance of adequate alveolar ventilation. This increased respiratory rate entailed an increase in dead space ventilation from 2,253 c.c. per minute to 7,875 c.c. per minute. The great burden which this situation constituted was not fully appreciated at the time. The patient died rather suddenly after talking with visitors. Autopsy failed to reveal a definite cause of death. The remaining lung tissue showed no congestion or edema to indicate heart failure, and the heart was normal. There was no clot in the pulmonary

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artery. The pleura over the left lung was thickened, and the underlying left lung contained many caseous nodules which proved to be typical of tuberculosis on section. The presumption is that the crippled and encapsulated left lung was barely able to carry the respiratory load postoperatively. The tidal volume was limited not only by the pain of the operated (right) side, but also by the pleural residuals on the left. We postulate that the diaphragm eventually became so fatigued that it would no longer respond with rhythmic contractions. It is felt that the increased production of carbon dioxide coupled with some crippling of the contralateral lung, the limitation of the tidal exchange, and a rather significant degree of paradoxical respiration associated with the five-rib thoracoplasty all contributed to the death. It is quite possible that a decrease in the volume of physiologic dead space (i.e., tracheotomy) might have allowed adequate alveolar ventilation to be accomplished with a great deal less effort. This is postulated because of the fact that the greatest abnormality found in the physiologic data was in the tremendous increase in the ventilation of the dead space.

DISCUSSION

Resectional surgery as a primary approach to the therapy of pulmonary tuberculosis is still in the developmental stage. The magnitude of the resection may vary from the removal of a small caseous nodule to a complete pneumonectomy. A great deal of work is currently being done in evaluating the efficacy of the various procedures as tools in overcoming tuberculosis. The present work is an attempt to determine the physiologic magnitude of each procedure, in order to aid in the selection of patients who are suitable for the various procedures. No consideration has been given to the therapeutic efficacy of the operations.

The most important principle involved in the modern surgical approach to pulmonary tuberculosis seems to be the extirpation of the significant disease without overexpansion of the remaining pulmonary tissue. Where the volume of tissue removed is small the problem of overexpansion is probably not important. But where as much as a lobe or major part thereof is to be removed, many surgeons and internists alike feel that there is an advantage in lessening the space which must be filled by the remaining lung tissue.

It was for this reason that a one-stage lobectomy and five-rib thoracoplasty was tried somewhat as a short cut to avoid two separate procedures.[2-4] The present study was undertaken in order to define the physiologic magnitude of this type of surgery and to compare it with the familiar effects of thoracoplasty.

All procedures which involve the removal of several ribs have one thing in common: i.e., the presence of a flail chest wall. The first paper in this series attempted to point out the disadvantage at which thoracoplasty places the respiratory mechanism. It was shown that tidal volume is limited immediately postoperatively (probably by pain and by the presence of paradoxical respiration). This necessitates an increased respiratory rate if alveolar ventilation is to be maintained. The maintenance of even the same rate of excretion of carbon dioxide is then more difficult than before surgery. The disadvantage against which the patient is working may be accentuated by the need for an increased rate of carbon dioxide elimination, such as occurs (1) with the acidosis which develops during general anesthesia, and (2) with postoperative fever.

The data herein presented reveal the physiologic magnitude of the combination of lobectomy and five-rib thoracoplasty to be significantly greater than

that of one stage of a seven-rib thoracoplasty. The increases in the metabolic rate, postoperative temperature, pulse rate, and carbon dioxide excretion were all significantly greater in the present series than in any stage of thoracoplasty alone. That the body labors under the same type of disadvantage following both procedures is shown by the limitation of tidal volume and tidal alveolar ventilation, and the great increase in respiratory rate and in dead-space ventilation.

The practical importance of these observations is obvious. It seems clear that the selection of patients who are suitable for the combination of resection and thoracoplasty must be rather carefully made. The mechanical and physiologic disadvantage at which the body is placed is very similar for the two procedures, probably because both involve the removal of several ribs with the resultant loss of respiratory efficiency. However, the magnitude of the effects associated with combined lobectomy-thoracoplasty is significantly greater than those associated with thoracoplasty alone, probably because of the greater surgical trauma associated with pulmonary resection per se.

It should be emphasized that the observations reported here were made after the subjects had accomplished complete correction of the respiratory acidosis which commonly occurs during thoracic surgical procedures. The need for eliminating the extra carbon dioxide which is frequently retained during general anesthesia may temporarily impose a very heavy load upon the crippled respiratory mechanism. An increase in the rate of respiration constitutes the only means by which the body can accomplish sufficient ventilation to achieve the elimination of any extra carbon dioxide. It is obvious that a patient with a greatly limited maximum breathing capacity and exercise tolerance before surgery might be unable to increase the rate and volume of ventilation sufficiently to meet the demands placed upon the respiratory system in the postoperative period. In the case of H. R. (reported above) there was a combination of pulmonary insufficiency, lung encasement, and paradoxical chest motion. It was only by great effort that he was able to maintain a passable alveolar ventilation. Finally, after the mild exertion of talking with visitors, the muscles responsible for carrying the extra work of respiration became fatigued and respiration ceased.

SUMMARY AND CONCLUSIONS

1. Eleven male patients undergoing an upper lobectomy in combination with a five-rib thoracoplasty were studied in the manner previously reported from this laboratory.

2. The inefficiency of the respiratory mechanism following lobectomy-thoracoplasty was significantly greater than that found immediately following each stage of thoracoplasty alone. Also, the magnitude of the stress placed upon the body by the combination of lobectomy and thoracoplasty was greater than that imposed by each stage of thoracoplasty alone.

3. There was one postoperative death in the present series, and this case is described in detail. The most significant features of the case were the great increase in the respiratory rate and in the dead-space ventilation and the de-

crease in tidal exchange. Autopsy revealed no cause of death, and it is concluded that the physiologic alterations found probably afford an adequate explanation for this postoperative death.

4. It is concluded, finally, that only patients with good respiratory reserve should be selected for a combination of thoracoplasty (which greatly cripples the efficiency of the respiratory mechanism) and pulmonary resection (which imposes a considerably greater load upon the body than does thoracoplasty alone).

The author is indebted to Dr. R. V. Ebert for much helpful advice, and to Shirley Lundgren and John Oganovich for technical assistance.

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THE INTRATHORACIC USE OF THE LARGE BOWEL AFTER SUBTOTAL ESOPHAGECTOMY FOR CANCER

Report of One Case

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THE surgical management of lesions of the esophagus has improved considerably in the last fifteen years because of advances in the field of modern anesthesia and the discovery of new antibiotic drugs. However, the reconstruction of the organ after resection, or its complete substitution when necessary, is a problem still challenging the skillfulness of all those who have tried to solve it.

The multiplicity of approaches to the esophagus, the variety of methods used, and the continuous appearances of new publications on the subject show that esophagoplasty has not yet come to its final stage of development, and that something is still unconquered by the surgeon. In reviewing these techniques we felt that perhaps the utilization of a segment of transverse and descending colons could give us a better chance to complete the operation begun by the esophagectomy, restoring the continuity to this part of the alimentary canal.

CASE REPORT

J. D. I. was a 51-year-old, white, married, Portuguese man, a baker. He came to the surgical clinic in November, 1950, complaining of retrosternal pain and progressive dysphagia for the last three months. He complained that he could not even drink water during the two days before admission. At the physical examination it seemed that he had lost much weight, but he still looked well. The blood pressure was 130/80. The fluoroscopic examination* revealed an obstruction localized between the upper and the middle thirds of the esophagus, showing irregular filling at the level of the aortic arch, with subtotal stenosis. Adequate preoperative care restored the blood cells to normal and the serum proteins to their level; the patient was considered in good condition for an operation. On Dec. 29, 1950, he was operated on under endotracheal general anesthesia (ether-oxygen mixture). Induction was carried out with sodium pentothal. The chest was entered through the left side, the sixth rib being resected in its whole extension (Fig. 1). Also the posterior segment of the fifth rib was resected. On exploring the mediastinum a tumor was identified in the esophagus at the level of the aortic arch. Further examination revealed this tumor spreading all over the organ to the diaphragm, but it did not invade the periesophageal tissues, thus permitting the dissection of the organ from the surrounding structures. Firm adhesions fixed the aorta and the esophagus at the level of the aortic arch. The diaphragm was then opened from its esophageal hiatus to the anterior abdominal wall and exploration was performed for metastasis in the liver and lymphatic nodes. As these structures were free from metastasis the stomach was sectioned from the esophagus just below the cardia and this opening closed with inverting cotton sutures in two planes. The lower end was closed with cotton in a purse-string fashion and the esophagus was easily isolated from the mediastinum to the aortic arch. At this point the adhesion was very firm, but it also came away. Palpation conducted over this area revealed that the tumor spread upward making it necessary to dissect the organ high into the neck.

From The Surgical Clinic Department, Instituto dos Comerciarios São Paulo.
Received for publication June 20, 1952.
*Performed by Dr. F. Bevilacqua.

As the patient was in a very poor general condition at this moment and the stomach was very small and retracted, it was felt that it would be difficult to bring it up to an anastomosis with the proximal end of the esophagus in the neck. As the dissection of the esophagus was completed up to the neck, it was left in the mediastinum, its lower end covered by a rubber finger, and the thorax was closed with nonabsorbable suture material by planes. The patient was then placed on his back and a gastrostomy of the Witzel type immediately performed; following an incision on the anterior border of the left sternocleidomastoid muscle the total thoracic esophagus was brought up to the neck and exteriorized (Fig. 1). The cervical esophagus also presented tumorlike nodules and for this reason it was resected, leaving only the first 3 or 4 cm. of the organ which were free of tumor masses. The proximal end of the organ was implanted in the skin of the neck where it formed a fistula. It must be emphasized that an attempt was made to remove all the visible periesophageal lymph nodes together with the organ while performing this subtotal esophagectomy for generalized cancer.

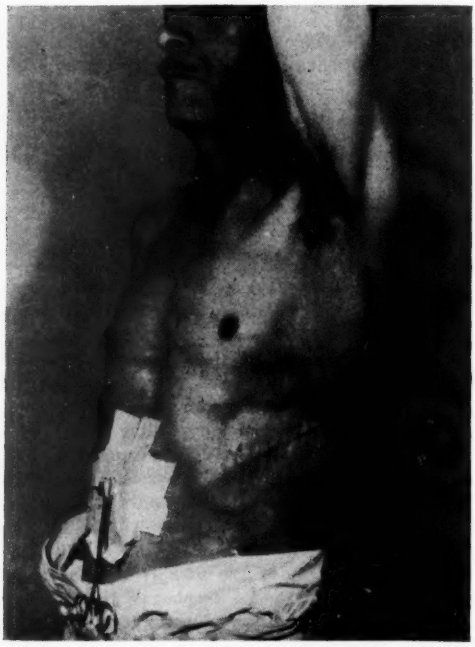

Fig. 1.—Patient showing incisions in the neck and chest; the cervical and epigastric fistulas.

A grayish fragment of this tumor tissue from the wall of the organ as well as a black lymph node were sent for pathologic examination.

Microscopically, the first of them showed an immature neoplasm of epithelial cells whose elements were grouped in irregular blocks exhibiting great polymorphism, as well as typical and atypical mitosis. The second material showed the structure of lymphatic nodular tissue substituted by connective proliferation presenting hyalinized points filled by anthracotic pigment and negative images of crystals together with small areas of neoplastic blocks of the type described. Diagnosis* was squamous carcinoma of the esophageal wall, carcinomatous metastasis in the lymph node, anthracosilicosis of the lymphatic gland.

*By Dr. Walter E. Maffei.

The early postoperative period was stormy, but in a few days the patient presented rapid and evident improvement in general condition. Fifteen days after the surgical procedure he had already gained some weight and three months later he had gained 13.2 pounds.

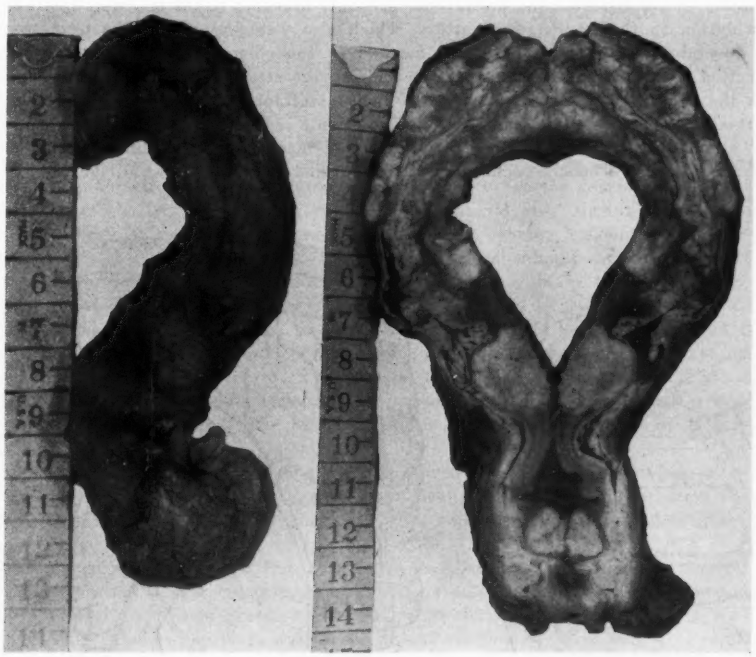

Fig. 2. Fig. 3.

Fig. 2.—Superficial aspect of the entire esophagus.
Fig. 3.—The esophagus opened through a longitudinal section showing the tumor along its surface.

Unfortunately the idea of eating by mouth had grown more and more in the patient and finally forced us to perform the second stage three months after the first stage, and therefore three months before the time was up, which had been hoped for as an observation period for possible metastasis. In March 31, 1951, after a convenient check-up of his condition, a reconstruction of the passage was attempted. In a right dorsal lateral position (45 degrees more or less) and under the same anesthetic mixture the patient was again operated on. Starting at the left paravertebral region an incision was made resecting the scar of the previous operation extending to the costal margin which was sectioned. This same incision was then extended downward to the abdominal anterior wall and parallel to the internal border of the left rectus muscle to the extent of three fingers below the umbilicus (Fig. 4). Both thoracic and abdominal cavities were opened, the diaphragm being sectioned in the same direction as before. It was decided to leave the feeble adhesions between the left lung and the thoracic wall to be freed just after the accomplishment of the abdominal stage.

On exploring the peritoneal cavity a retracted stomach was found, strongly adherent by the previous gastrostomy to the anterior abdominal wall and also to the gastrohepatic ligament. There were found in this last structure some hard and grayish masses, which

were dissected and proved to be carcinomatous metastases. There were no suspected nodules in the mediastinum.

The great omentum was then isolated from the transverse colon and its connections to the splenic angle. Afterward the transverse and descending mesocolons were explored and their arterial circulation studied carefully. The middle colic artery was easily identified by its diameter and preserved. All other branches to the left were ligated to its inoseulation to the first sigmoid artery, which also was preserved. The next step was the dissection of the transverse and descending mesocolons in their entirety. In this dissection care was taken to preserve the middle colic artery and all its branches together with the isolated loop that we wanted to free, maintaining in this way its blood supply. By this procedure we got a long and movable colonic segment attached to the abdomen by only a long mesocolic pedicle containing the middle colic artery (Fig. 5). This pedicle reached the isolated colonic segment near its proximal extremity (that is, the right end of the isolated loop).

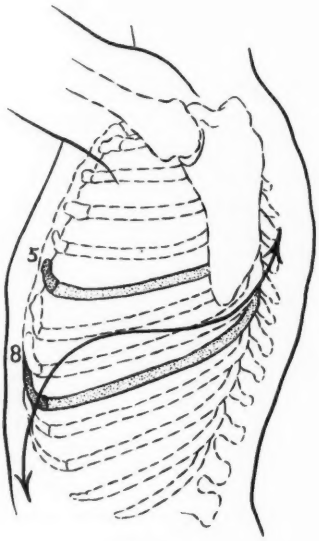

Fig. 4.

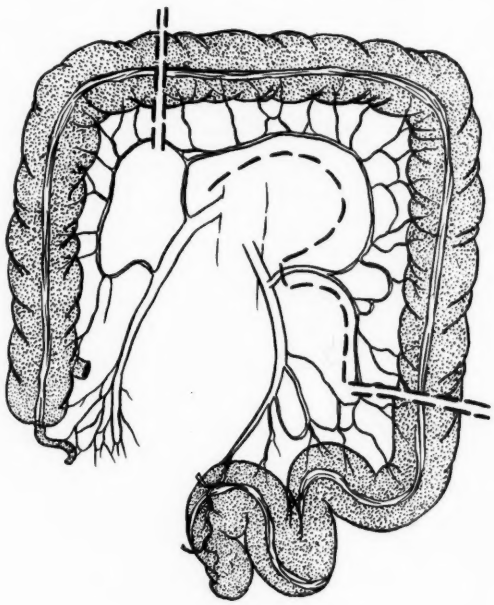

Fig. 5.

Fig. 4.—Line of incision utilized in the attempt to perform an intrathoracic esophagoplasty.

Fig. 5.—Diagrammatic sketch showing the segments of isolated colon used in the chest and the circulation used.

The colonic continuity was re-established by an end-to-end continued suture with chromic catgut, using the Vasconcellos clamp. A second plane using cotton stitches was done over the first one. The opening in the mesocolon was closed completely, then the proximal end of the isolated colonic segment was anastomosed to a new opening made at the highest point of the gastric wall by continuous chromic catgut suture in two planes. Careful attention was given to the arterial mesocolic pedicle which occupied a better position posteriorly to the stomach where it was not exposed to any traction. Also care was taken not to twist this isolated loop to the stomach. After his cologastric anastomosis was ready, the stomach was brought up as far as possible to the esophageal hiatus of the diaphragm now wide open and fixed to the borders of this muscle, with the intention of

preventing any traction on the cologastric suture. This maneuver permitted a small portion of the stomach, containing cologastric anastomosis, to remain supradiaphragmatically. Sufficient room was left between the stomach and the border of the diaphragm for passage without constriction of the arterial pedicle going to the isolated segment. In order to avoid any possible damage to this pedicle, a splenectomy was carried out. The left pleural space was then freed of its adherence to the left lung and the isolated segment was conducted and placed in a good position to the internal side of the posterior thoracic wall near the mediastinal aspect, where it was fixed by anchoring stitches. The diaphragm was then completely closed to the most anterior portion. The abdomen was also closed in planes using cotton interrupted suture.

A left cervical incision over the anterior scar was made and all the elements were incised and retracted until a plane could be reached posterior to the left clavicle. Through the left transthoracic opening a connection was tried between the cervical region and the highest part of the left thoracic cavity, close to the middle line. During this procedure a large retroclavicular venous blood vessel was severed, producing a hemorrhage which was controlled with great difficulty. As the patient entered hemorrhagic shock we were obliged to alter plans for this intervention, and instead of making the cervical anastomosis between the esophagus and the colon at this stage we decided to implant the colon to the anterior thoracic wall. The distal end of the isolated colonic loop was fistulized to an opening made in the left, second intercostal space, two fingers from the sternal border. It was sutured by interrupted cotton stitches in two planes, one muscular and the other cutaneous. Penicillin and streptomycin were placed in the left cavity which was closed by planes without drainage. The left cervical incision was then closed by interrupted cotton sutures.

In summary, an attempt was made to perform an intrathoracic reconstruction of the esophagogastric continuity by using antiperistaltically a segment of transverse and descending colons after a subtotal esophagectomy for carcinomatosis of the esophagus (Figs. 2 and 3).

Even though this patient was able to re-establish his blood pressure seven hours after the operation a new hemorrhage at the same point brought him again into shock. Treated correctly his blood pressure returned to its normal level. However, forty-eight hours after the intervention there was a very serious overflow of saliva and other secretions to the tracheobronchial tree. Two bronchoscopic aspirations were necessary to restore his general condition to normal once more. At the end of the third postoperative day he had the first spontaneous bowel movement, with a second one during the night.

Also the intrathoracic loop started to move, beginning by eliminating fecaloid fluid material and later gastric contents. By introducing a tube into the chest opening, this loop could be washed of its contents with saline solution. After this procedure it was noted that any fluid introduced into the loop could be seen appearing spontaneously through the gastrostomy some time later demonstrating that this loop was functioning toward the stomach.

The general condition of this patient improved rapidly, and he reported he felt much better. In the afternoon of the eighth postoperative day following a severe cough, there was dehiscence of the abdominal part of the surgical wound, through which jejunal loops were protruding. Developing shock, he was again taken to the operating table and under general anesthesia all the protruding jejunal loops were put back into the peritoneal cavity. The examination of the peritoneal cavity made at this moment did not reveal abnormality or any sign of dehiscence of the colocolic anastomosis whatever. However, a certain amount of serofibrinous exudate was found in the cavity and was attributed to the prolonged evisceration of the loops. Penicillin and streptomycin were placed in the peritoneal cavity before it was closed by separated cotton stitches. Once more the patient came out of shock and there was an improvement in his general condition. All the thoracic symptoms and signs indicated a better local condition. This was confirmed by several x-ray pictures taken of the patient in bed during this postoperative period.

Suddenly on the night of the eleventh day he felt ill and died. The autopsy report, performed at the Institute Adolpho Lutz, presented the following main points.

General Data.—A white, adult man presented three cutaneous fistulas localized as follows: The first, on the anterior border of the sternocleidomastoid muscle, two fingers above the internal end of the left clavicle; the second, two fingers from the left sternal border, in the second interspace; the third in the epigastric region, in the middle line, where a rubber tube can be seen. Three surgical incisions were noted: The first corresponding to the first fistula along the muscle; the second already healed in the middle line, xipho-umbilical, where there was a third fistula. The third surgical scar was recent and irregular, going from the left paravertebral space to the anterior aspect of the chest forming an arch over the left sixth rib, to the costal margin and downward over the anterior rectus muscle to a point inferior to the umbilicus. This last incision presented cotton stitches over its line. By opening this incision it was noticed that the sixth rib had been removed surgically. Both chest and abdominal cavities were then open and only separated by the diaphragm. In the abdomen it was noticed that a serofibrinous exudate extended over the cavity. In the left side of the abdomen there was a fecaloid material exuding from a dehiscence in the line of suture of the end-to-end anastomosis between the right part of the transverse colon and the sigmoid colon. It was still noted that the stomach was attached to the anterior abdominal wall, where there was a gastrostomy. The highest part of the stomach was sutured and surrounded by the diaphragm entering into the thorax to some degree. By cutting the stitches on the diaphragm it was observed that a pedicle of the mesocolon entered the chest cavity behind the stomach. On opening the diaphragm, a dark fluid was emitted from the left pleural cavity to the abdomen. The transverse and descending colons were in the left pleural cavity extending from the second, left interspace to the highest part of the stomach. Both the superior colonic extremity as well as the cologastric anastomosis showed good healing. There was no spleen (removed).

Diagnosis.—Generalized peritonitis due to dehiscence of anastomatic line of the end-to-end colocolic suture. Gastrostomy (operation). Colostomy in the second left interspace (operation). Supradiaphragmatic cologastrostomy in the left pleural space (operation). Fistula of the esophagus on the left side at the base of the neck (operation). Splenectomy (operation).

Description of Organs Involved.—Esophagus: It is noted that the esophagus was turned toward the cutaneous fistula in the neck and that the esophagus was reduced to only 3 or 4 cm. of its upper third. Thoracic cavity: The cardiac area was normal; pleural membranes were free. The left pleural space exhibited approximately 500 Gm. of dark fluid. Right lung: Form and size were normal. Crepitation was present. The sagittal section was dark red in color, emitting when compressed a small amount of blood (slight congestion). Left lung: The size was small due to collapse of the lower lobe. Cut surface was normal. Heart: No information worth noting.

Causa Mortis.—Toxemia and generalized peritonitis due to dehiscence of suture line in the intestinal anastomosis.*

COMMENTS

The main objective of this paper is the presentation of a case of generalized carcinoma of the esophagus treated by a subtotal esophagectomy where we had to face the problem of a plastic reconstruction of the organ.

It was clear from the beginning of the surgical treatment that this patient could not expect to have a long survival due to metastasis already observed at the first stage of the operation. However, his will to live and to eat again

*Dr. Pedro Gherardi, Jr.

through his mouth had forced us to break into our observation period three months from the first operation and try a re-establishment of the continuity of the upper digestive tract. Considering the conditions revealed at the time of the esophagectomy, we had no doubts about the unfeasability of bringing his stomach up to the neck, as was our intention at first. Besides its small size, the stomach should be expected to be firmly adherent and fixed to the anterior abdominal wall at the point where the previous gastrostomy was done.

Upon analyzing the advantages and disadvantages of the different methods in a review of the surgical literature and after reading Orsoni's paper[6] a few days before the operation, we decided on an intrathoracic colonic substitution of the esophagus as perhaps the best chance we could give to our patient, even though it was observed that the colon had never been used for this type of plastic surgery until this time. We also were, and still are, favorable to the one-stage resection with an esophagogastric anastomosis described by Sweet, Garlock, and many others, but we are sure this procedure can be useful in cases where the lesions, whatever they may be, are located very high and the stomach cannot be brought to that point.

REVIEW OF LITERATURE ON INTRATHORACIC ESOPHAGOPLASTY

Although prethoracic esophagoplasty has had very satisfactory results, many authors since the beginning of the century have tried to obtain an intrathoracic esophagoplasty. Attempts were made by Wullenstein (1904), Sauerbruch (1905), Willy Meyer (1913), Marwedel (1913), Enderlen and Hotz (1914) and others to perform this intervention on an experimental basis. Lorthioir and his associates tried to establish an anastomosis to the jejunum after the resection of the diseased segment of the esophagus. Kümmel[1] was the first to use an intrathoracic approach by the Kirschner method, doing an anastomosis between the esophagus and the stomach freed from its vascular connections. However, the majority of these interventions were performed in cases of esophageal stenosis due to lye strictures. Tumorous cases were considered very dangerous and useless.

Many tissues and organs have been used up until the present time to substitute for the diseased esophagus, for example, the skin, epidermis, fascia lata, as well as different parts of the digestive tract (stomach, jejunum, and colon).

In 1946, Rienhoff[2] was the first to use jejunum intrathoracically. He made the anastomosis of a free proximal jejunal loop antiperistaltically to the esophagus at the level of the aortic arch (Fig. 6). Bricker, Burford, and Eiseman[3] related in 1949 the re-establishment of the upper thoracic esophagus resected by using a tubed pedicle graft obtained from the cervical regions, which was introduced into the upper thorax and then anastomosed to the lower esophagus Fig. 6).

At the same time, Harrison[4] described the technique used by him in two cases. He re-established through an end-to-end suture the transit of an esophageal segment resected previously using an isolated jejunal loop. This loop was sutured to the esophagus in the neck and below to the gastric fundus completing an isoperistaltic intrathoracic esophagoplasty (Fig. 6).

In November, 1950, Robertson and Sarjeant[5] published a paper on the reconstruction of the esophagus in one stage by using an isoperistaltic segment of the jejunum through an undermined tunnel in the anterior mediastinum (Fig. 6); from the technical surgical point of view, the descriptions of the methods devised by Bricker and associates, Harrison, Robertson, and Sarjeant are excellent and should be read by those considering all these procedures.

In March, 1951, Paul Orsoni presented a study on the utilization of the transverse and descending colons for the intrathoracic plastic reconstruction of

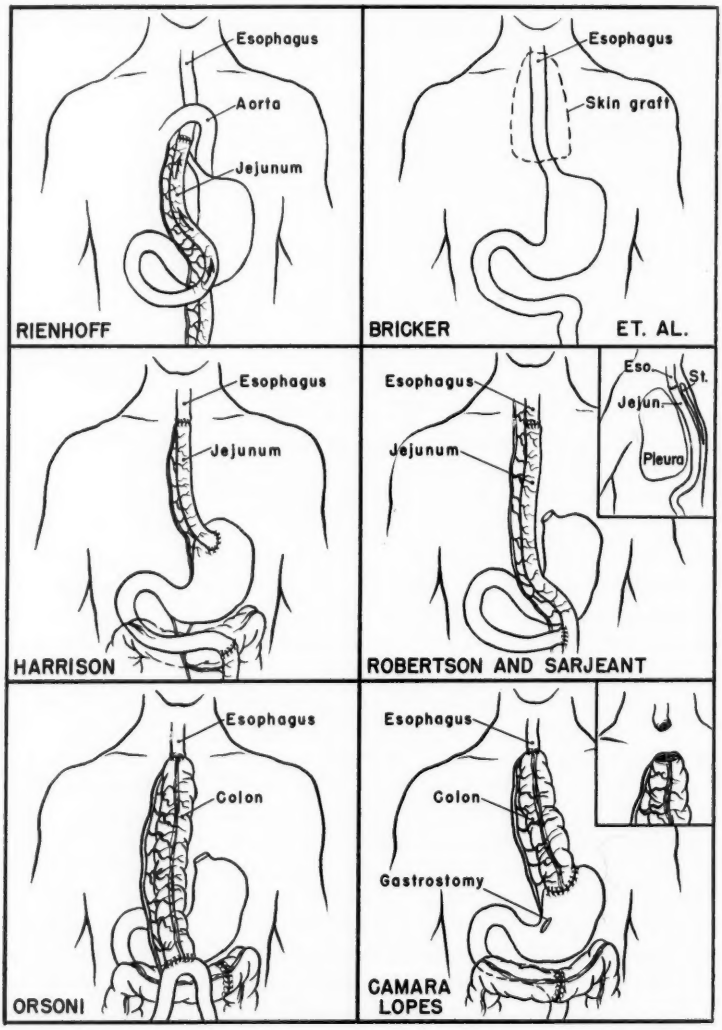

Fig. 6.—The different types of intrathoracic esophagoplasties.

the esophagus. The reasons for the use of this organ were studied by Lemaire, who performed several arteriographies on the colic arteries. Orsoni considered the thoracic use of the colon most advisable. Together with Lemaire (Fig. 6) they have anastomosed an isolated colonic segment between the left portion of the resected esophagus and the first jejunal loop in an attempt to avoid any possible gastric regurgitation to the esophagoplasty so performed.

DISCUSSION

While most of our experience could be looked upon as too limited we feel that this procedure is worth describing and should be kept in mind by others as something else to add to the resources useful to those who have to handle lesions of the upper esophagus.

In reviewing the articles published by different authors on the pre- and intrathoracic esophagoplasty and the results heretofore obtained with them, we have come to the following conclusions.

1. According to the recent works of Toupet and Lemaire, colonic circulation is better than jejunal and can be considered quite sufficient to maintain an isolated colonic segment alive and in good condition, regardless of the length of this segment.

2. Transverse colon can be used iso- or antiperistaltically as has been demonstrated in the cases related by Kelling and Vulliet.

3. According to Reagan and Puestow, working with isolated segments, the colon still keeps its responses to certain drugs, although it loses its mesenteric innervations, a fact that cannot be reproduced in the jejunum.

4. Experiments performed by several authors (Moroney, Flory, Harding, Ivy and co-workers) proved the colon to be more resistant to digestive secretions than jejunum, therefore being much more applicable for its use in esophagoplasty.

5. From the anatomical and functional point of view it is more advisable to perform an esophagoplasty between the upper part of the cervical esophagus or pharynx and the stomach, than to make an anastomosis of its lower extremity to the jejunum as was done by Rienhoff, Robertson and Sarjeant, and Orsoni and Lemaire, keeping in this way the gastric digestive function.

6. The colon is more resistant and less sensitive than jejunum.

7. Much smaller than the stomach in diameter, the colon occupies a smaller space inside the thorax, leaving the stomach with its normal functions with regard to capacity and digestion.

8. The use of a colonic loop intrathoracically gives a better esthetic appearance to the individual and protects the loop.

9. Intrathoracic colonic esophagoplasty has the advantage of allowing any possible treatment on the diseased esophagus, extending the indications of this procedure to the treatment of some of the congenital lesions, and also to the malignant tumors of the organ.

SUMMARY

1. A case of generalized carcinoma of the esophagus presenting only a small mucous lesion at the level of the aortic arch is described.

2. A subtotal esophagectomy was performed without the possibility of the re-establishment of an esophagogastric continuity in one single operation.

3. Three months later an intrathoracic esophagoplasty was attempted by using a segment of the large bowel to re-establish the transit.

4. A careful review of the literature has not revealed a description of this intrathoracic technique in man.

5. This plastic operation may be considered feasible and more physiologic than esophagoplasties described up to the present time.

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THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Thirty-Third Annual Meeting

March 27, 28, and 30, 1953, Fairmont Hotel

San Francisco, California

Program

Friday Morning, March 27, 1953

9:00 A.M. Business Meeting

9:30 A.M. Scientific Session

1. **Unilobar Obstructive Emphysema in Infancy Treated by Lobectomy.**
   Herbert Sloan, Ann Arbor, Mich.
2. **Mucocele, Congenital Bronchiectasis, and Bronchiogenic Cyst.**
   B. H. Ramsay, and Francis X. Byron, Los Angeles, Calif.
3. **Hydatid Disease as It Affects the Thoracic Surgeon.**
   M. P. Susman (by invitation), Sydney, Australia
4. **Surgical Considerations in Focalized Pulmonary Histoplasmosis.**
   James H. Forsee, and Thomas F. Puckett (by invitation), Denver, Colo.
5. **Extensive Bronchiectasis: A Clinical and Physiologic Evaluation of the Results of Surgical Treatment.**
   Edgar P. Mannix, Jr. (by invitation), Francis S. Gerbasi (by invitation), Charles E. O'Brien (by invitation), and Richard H. Adler (by invitation), Forest Hills, N. Y.
6. **Decisions in Thoracic Surgery as Influenced by Knowledge of Pulmonary Physiology.**
   Warriner Woodruff, Carl G. Merkel, and George W. Wright, Saranac Lake, N. Y.

Friday Afternoon, March 27, 1953

2:00 P.M. Scientific Session

7. **Localization of Radioactivity in the Lung and Thoracic Lymph Nodes.**
   J. Ray Bryant (by invitation), and Harold F. Berg (by invitation), Louisville, Ky.
8. **The Ability of the Pulmonary Vascular System to Influence the Spread of Tumor Emboli.**
   Edwin A. Lawrence, Donald B. Moore (by invitation), and George I. Bernstein (by invitation), Indianapolis, Ind.
9. **Bronchial Resection and Anastomosis.**
   Paul W. Gebauer, Honolulu, T. H.
10. **Bronchiolar ("Alveolar Cell") Carcinoma of the Lung.**
    Clifford F. Storey, K. P. Knudtson (by invitation), and B. E. Lawrence (by invitation), St. Albans, N. Y.
11. **The Significance of Pulmonary Hypertension as a Cause of Death Following Pulmonary Resection.**
    W. E. Adams, John F. Perkins, Jr. (by invitation), and Adolfo Flores (by invitation), Chicago, Ill.

12. **Prevention of Chest Wall Defects: Use of Tantalum and Steel Mesh.**
Donald B. Effler, Cleveland, Ohio

Saturday Morning, March 28, 1953

9:00 A.M. SCIENTIFIC SESSION

13. **The Present Chaos Which Exists Regarding Pulmonary Resection.**
E. J. O'Brien, and Paul V. O'Rourke, Detroit, Mich.

14. **The Use of Isoniazid as a Prophylactic Antibacterial Agent in Pulmonary Resection for Tuberculosis: A Preliminary Report.**
Max E. Childress (by invitation), Mildred E. Thoren (by invitation), and Albert C. Daniels (by invitation), Weimar, Calif.

15. **The Influence of Long-Term Chemotherapy on the Surgery of Pulmonary Tuberculosis.**
John D. Steele, B. G. Narodick (by invitation), and A. V. Cadden (by invitation), Milwaukee, Wis.

16. **Segmental Resection of Pulmonary Tuberculosis: An Analysis of Three Hundred Cases Followed From One to Five Years.**
J. Maxwell Chamberlain, Robert Klopstock, and Charles F. Daniels (by invitation), New York, N. Y.

17. **Electrolyte Studies in Pericardial Resection.**
Walter B. Crandell, Andrew Yeomans (by invitation), David Hoffman (by invitation), and George H. Stueck, Jr. (by invitation), White River Junction, Vt.

18. **Bronchial Closure in Pulmonary Resection: A Clinical and Experimental Study Using a Pedicled Pericardial Fat Graft Reinforcement.**
Lyman A. Brewer, III, Frank S. Dolley, Edward L. King (by invitation), and Angel F. Bai (by invitation), Los Angeles, Calif.

Saturday Afternoon, March 28, 1953

2:00 P.M. EXECUTIVE SESSION

3:00 P.M. SCIENTIFIC SESSION

ADDRESS OF THE PRESIDENT—Robert M. Janes, Toronto, Ont.

19. **The Experimental Closure of Atrial Septal Defects.**
Harris B. Shumacker, Jr., and Thomas C. Moore (by invitation), Indianapolis, Ind.

20. **Surgical Treatment of Mitral Insufficiency: An Experimental Study.**
Max G. Carter, and John M. Gould (by invitation), New Haven, Conn.

21. **Pulmonary Valvuloplasty Under Direct Vision: The Aid of a Mechanical Right Heart.**
F. D. Dodrill, Robert A. Gerisch (by invitation), Aran S. Johnson (by invitation), and Edward Hill (by invitation), Detroit, Mich.

6:30 P.M. COCKTAILS AND DINNER—Fairmont Hotel.

Attendance limited to members of the Association and their wives, invited speakers and their wives. Dinner dress.

Monday Morning, March 30, 1953

9:00 A.M. SCIENTIFIC SESSION

22. **The Problem of the Hiatus Hernia of the Diaphragm.**
Conrad R. Lam, and Leo J. Kenney (by invitation), Detroit, Mich.

23. **Muscle Wall Tumors of the Esophagus.**
Richard H. Sweet, and Lamar Soutter, Boston, Mass.

24. **Reinforcement of Aortic Aneurysms by Wrapping: A Research Report.**
J. Karl Poppe, Portland, Ore.

25. **Clinical and Cytologic Changes in Patients Receiving Aerosol Trypsin and Desoxyribonuclease.**
Seymour M. Farber (by invitation), David A. Wood (by invitation), R. Daniel Gorman (by invitation), and Samuel Pharr (by invitation), San Francisco, Calif.

26. **Torsion Ballistocardiography in Cardiovascular Surgical Patients.**
C. Frederick Kittle (by invitation), Kurt R. Reissmann (by invitation), E. Grey Dimond (by invitation), and Paul W. Schafer, Kansas City, Kan.

27. **An Experimental Study of the Effect of Parasympathetic Denervation of the Lung on Pulmonary Artery Pressure.**
Victor H. Kaunitz (by invitation), and Murray N. Andersen (by invitation), Buffalo, N. Y.

Monday Afternoon, March 30, 1953

2:00 P.M. SCIENTIFIC SESSION

28. **The Role of the Bronchial Artery Circulation in the Etiology of Pulmonary and Pericardial Suppuration: An Experimental Study.**
Richard S. Hahn (by invitation), and Emile Holman, San Francisco, Calif.

29. **The Production and Repair of Interatrial Septal Defects Under Direct Vision With the Assistance of an Extracorporeal Pump Oxygenator Circuit.**
Bernard J. Miller (by invitation), Victor F. Greco (by invitation), Burgess A. Smith (by invitation), and John H. Gibbon, Jr., Philadelphia, Pa.

30. **The Oxygenator Pump in Total By-Pass of Heart and Lungs: Laboratory Evaluation and Clinical Use.**
James A. Helmsworth, Leland C. Clark, Jr. (by invitation), Sam Kaplan (by invitation), Roger T. Sherman (by invitation), and Thomas Largen (by invitation), Cincinnati, Ohio

31. **Experiences With Cardiopexy in the Treatment of Coronary Disease.**
Samuel A. Thompson, New York, N. Y.

32. **Cardiac Surgery Under Hypothermia.**
Charles P. Bailey, Brian Cookson (by invitation), William Likoff (by invitation), H. E. Bolton (by invitation), D. F. Downing (by invitation), and W. B. Neptune (by invitation), Philadelphia, Pa.

33. **The Management of the Dangerously Torn Auricle.**
William K. Swann (by invitation), Thomas L. Lomasney (by invitation), and Jorge Rodrigues-Arroyo (by invitation), Knoxville, Tenn.

PAPERS TO BE READ BY TITLE

1. **One-Stage Thoracoplasty Using the Trapp Adhesive Hemicast.**
Frederick L. Coddington (by invitation), Mt. Edgecumbe, Alaska

2. **A Comparison of Thoracoplasty and Resection Therapy in the Treatment of Pulmonary Tuberculosis.**
Thomas G. Baffes (by invitation), Phyllis Schlotterbeck (by invitation), and Paul T. De Camp, New Orleans, La.

3. **Replantation and Transplantation of Lobes of the Lung of Dogs.**
   Felix A. Hughes, Jr., John W. Polk (by invitation), Francis E. Foley (by invitation), and John R. Fox (by invitation), Memphis, Tenn.
4. **Clinical Evaluation of Cortisone, Tyral, and Intermittent Positive Pressure Breathing (IPPB) Therapy in Thoracic Surgery by Lung Function Studies.**
   George D. Boone (by invitation), Tucson, Ariz.
5. **Pectus Excavatum: Report of Five Additional Cases Treated Successfully With a Rib Strut.**
   Ralph A. Dorner, Des Moines, Ia.
6. **Esophagophrenic Syndrome.**
   Reuben I. Shapiro (by invitation), Detroit, Mich.
7. **Circulatory Status of Resected Tuberculous Pulmonary Lobes.**
   Raymond J. Barrett (by invitation), Mikio Masaki (by invitation), and J. C. Day, Detroit, Mich.
8. **Bronchovascular Variations in the Normal Lung.**
   W. E. Bloomer (by invitation), A. A. Liebow (by invitation), and M. R. Hales (by invitation), New Haven, Conn.
9. **Asymptomatic Lung Cancer.**
   John L. Pool, William L. Watson, and Henry D. Diamond (by invitation) New York, N. Y.
10. **Physiologic Observations Before and After Decortication of the Lung.**
    Walter G. Gobbel (by invitation), Allan Stranahan, and Ralph D. Alley (by invitation), Albany, N. Y.